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(Continuing the Journal of the Allied Dental Societies)

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The effort to issue the numbers of the *Journal* promptly on the new schedule of bi-monthly publication, and the extra labor resulting from the expansion of the present volume, prevented special effort to increase the Endowment Fund, to which there have been no recent additions. The annual deficit, larger than usual becaue of the increased bulk of this Volume, will be liquidated as heretofore by supporters of the cause of professional journalism.

PROCEEDINGS OF AFFILIATED DENTAL SOCIETIES PUBLISHED AS INDEPENDENT JOURNALS WITHIN THE JOURNAL OF DENTAL RESEARCH

The dental societies named on the title page publish their proceedings on conditions that make each society's group of papers, in effect, its own independent journal within the *Journal of Dental Research.* Papers contributed by societies are controlled and edited by the respective associations through their official representatives. Reprints of sections may be obtained for issuance in separate volumes of transactions. See the title page of this Volume, and compare it with those of Volumes V, VI, VII, and VIII.

"DENTISTRY" AND "STOMATOLOGY"

This *Journal* uses the term "dentistry" to signify the science and art of oral health service in all its local and systemic relationships. The meanings of words, which are not necessarily restricted to *etymological* significance, acquire expanded or new import in accordance with convenience and usage. Thus the term "medicine," which originally meant to remedy or cure, now also means *to make remedial or curative treament unnecessary.* "Stomatology" is a synonym for "dentistry." We prefer the older term because its use respects both the history of dentistry and the efforts of those who have brought dentistry to its present status.

In Memoriam

Robert Robbin Andrews	1921, iii, page 2
George Andrew Bates	1928, viii, page 2
Edward Augustus Bogue	1928, viii, page 553
Clarence Jones Grieves	1928, viii, page 325
Holmes Condict Jackson	1928, viii, page 117
William Jarvie	1922, iv, page xxii
Newell Sill Jenkins	1919, i, page 416; 1920, ii, page 186
Samuel James Meltzer	1920, ii, page 534
Frederic austin Peeso	1929, ix, page 2
Frank Thorn Van Woert	1927, vii, page 347

CONTENTS

No. 1, February

Frederic Austin Peeso. *Arthur N. Cross*..... 3

A Critical Analysis of the Dental Literature Dealing with the Effects of Dietary Variations upon the Structure of Teeth. *Henry Klein*, D.D.S.... 5

The Relation of Nutrition to General Immunity. *Sherman L. Davis*, M.D., A.B., A.M., Ph.D..... 11

Discussion of the paper by Davis. *Arthur F. Coca*, M.D., *Arthur H. Merritt*, D.D.S., *Leuman M. Waugh*, D.D.S., *Leland Barrett*, D.D.S., *Joseph D. Eby*, D.D.S., *Henry W. Gillett*, D.M.D., *Charles F. Bödecker*, D.D.S., and *Dr. Davis*.. 20

Biochemical Studies of Streptococci. I. A Comparative Study of Some Common Media for the Cultivation of Streptococci. *Frances Krasnow*, Ph.D., and *William J. Gies*, Ph.D., F.A.C.D..... 29

The Bacterial Destruction of Dental Enamel. *Charles F. Bödecker*, D.D.S., F.A.C.D., and *Henry W. C. Bödecker*, B.S., D.D.S., M.D., D.M.D. (Ger.)..... 37

A Study of Some of the Materials Used in the Casting Process. *Oskar Solbrig*, D.D.S..... 55

No. 2, April

International Association for Dental Research. Scientific Proceedings of the Ann Arbor Section. *U. G. Rickert*, A.M., D.D.S., *Editor*..... 75

International Association for Dental Research. Scientific Proceedings of the Chicago Section. *B. Orban*, M.D., *Editor*..... 79

Painful Sockets after Extractions. A Preliminary Report on the Investigation of Their Etiology, Prevention, and Treatment. *Joseph Schroff*, M.D., D.D.S., and *Henry A. Bartels*, B.S., D.D.S..... 81

Medico-dental Case Records: Seventh Report. A Clinico-pathological Study. *Bissell B. Palmer, Jr.*, D.D.S., F.A.C.D., and *Malcolm W. Carr*, D.D.S.. 89

Dental Education: A Factor in Public Welfare. *William J. Gies*, Ph.D., F.A.C.D., and *James Sullivan*, Ph.D..... 107

Biochemical Studies of Streptococci. II. Availability of 671 Synthetic Media. *Frances Krasnow*, Ph.D., and *Margaret L. Rosenberg*, M.A..... 123

Studies of the Validity of Advertised Claims for Dental Industrial Products. II. Lymeogene, a French Toothpowder. *Research Council of the New York Academy of Dentistry*, with the collaboration of *Charles F. Bödecker*, D.D.S., F.A.C.D..... 171

The Face and Teeth of Man. A Study of Growth and Position. *Milo Hellman*, D.D.S., F.A.C.D..... 179

Bone Changes and Their Significance. *Egon Neustadt*, M.U.D. (Vienna)..... 203

Tissue Reaction in the Apical Region to Known Types of Treatment. *James Roy Blayney*, B.S., M.S., D.D.S..... 221

Discussion of the paper by Blayney. *E. G. Van Valey*, D.D.S., and *Dr. Blayney*... 242

The Biological Concept of Partial Prosthesis, and the Technic for a Fixed Bridge that Complies with it. *W. G. Bridge*, D.M.D..... 251

No. 3, June

International Association for Dental Research. Scientific Proceedings of the Seventh Annual Meeting, Chicago, March 23 and 24, 1929. Compiled by *William J. Gies*, F.A.C.D., *Secretary*..... 269
Mechanism of Intoxication by Intestinal Absorption in Chronic Oral Septic Foci. *Lloyd Arnold*, M.D., and *C. W. Stuart*, D.D.S..... 305
Biochemical Studies of Streptococci. III. Availability of Carbon Compounds. *Frances Krasnow*, Ph.D., and *Margaret L. Rosenberg*, M.A..... 321
Oral Microörganisms of the Dog. *Theodore B. Beust*, M.D., D.D.S., F.A.C.D.... 333
Micro-cinematography in Medical Research. *Heinz Rosenberger*, C.E..... 343
A Saddle, Physiological in Construction and Design, for Precision Attachments. *Anna Rosa Bruenn*, D.D.S..... 353
Bone Changes Induced by Periodontic Treatment. *S. Sorrin*, D.D.S..... 359
Biometrics of Identical Twins from the Dental Viewpoint. *Samuel Goldberg*, M.S., L.D.S., D.D.S..... 363
How Dental Statistics Are Secured in the Metropolitan Life Insurance Company. *Thaddeus P. Hyatt*, D.D.S., F.A.C.D. Addendum: *Alfred J. Lotka*, M.A., Sc.D..... 411

No. 4, August

A Comparison of Clinical, Roentgen, and Microscopical Findings in Fifteen Cases of Infected Vital Pulps. *Kurt H. Thoma*, D.M.D..... 447
Lymph Channels in Dentine and Enamel Stained by Amalgam. *Edmund Applebaum*, D.D.S..... 487
A Case of Heterotopic Ossification in an Untreated Granuloma. *Lester Richard Cahn*, D.D.S..... 503
A Study of the Efficiency of Dental Fillings. *C. Jane Fraser*, M.A..... 507
The Germicidal Effects of the Ultraviolet Rays on Staphylococcus Aureus. *Dewet Vorster*, L.D.S., R.C.S., D.D.S., M.S..... 519
A Preliminary Report on the Relation of Dental Aciduric Bacteria to Lactobacillus Acidophilus. *Theodor Rosebury*, D.D.S., *Richard W. Linton*, Ph.D., and *Leon Buchbinder*, A.M..... 529
Biochemical Studies of Streptococci. IV. Availability of Nitrogen Compounds. *Frances Krasnow*, Ph.D., *Benjamin Harrow*, Ph.D., and *Miriam Reiner*, A.B..... 531
Problems in Teaching Pre-Clinical Subjects, with Special Reference to the Preparation of the Student for Research. *Isaac Schour*, B.S., D.D.S., M.S..... 545
The Historical Significance of Professor Gies' 1927 Study in the Stomatologic Movement. *Alfred J. Asgis*, Sc.B., M.A., D.D.S..... 555
Preliminary Comment on Some Statements in Dr. Asgis' Report on the Carnegie Foundation's Study of Dental Education. *William J. Gies*, Ph.D.... 595

No. 5, October

International Association for Dental Research. Scientific Proceedings of the Chicago Section, May 23, 1929. *B. Orban*, M.D., Editor..... 609

PREVENTIVE DENTISTRY. A SYMPOSIUM. *William Dwight Tracy*, D.D.S., F.A.C.D.; *Harold J. Leonard*, D.D.S., B.A.; *Frank A. Delabarre*, D.D.S., M.D., F.A.C.D.; and *Sherman L. Davis*, M.D., A.M., Ph.D. 611

THE PENETRATIVE POWER OF THE BACTERICIDAL RAYS IN THE ULTRAVIOLET PART OF THE SPECTRUM, AS EMITTED BY CARBON C. *DeWet Voster*, L.D.S., R.C.S., D.D.S., M.S. 641

BIOCHEMICAL STUDIES OF STREPTOCOCCI. V. AVAILABILITY OF INORGANIC SUBSTANCES. *Frances Krasnow*, Ph.D., and *Miriam Reiner*, A.B. 653

A PHYSICO-CHEMICAL STUDY OF THE STRUCTURE OF DENTAL ENAMEL. *Henry Klein*, D.D.S., and *William R. Amberson*, Ph.D. 667

THE FATE OF THE AMELOBLASTIC CELLS OF THE ENAMEL ORGAN. *B. G. Anderson*, D.D.S. 689

THE PENETRATION OF THE TISSUE BY VINCENT'S ORGANISMS. *Lester Richard Cahn*, D.D.S. 695

EARLY HUMAN TOOTH DEVELOPMENT, WITH SPECIAL REFERENCE TO THE RELATIONSHIP BETWEEN THE DENTAL LAMINA AND THE LIP-FURROW BAND. *Isaac Schour*, B.S., D.D.S., M.S. 699

No. 6, DECEMBER

DEMONSTRATION OF ANAPHYLACTIC RESPONSE IN HYPERSUSCEPTIBLE GUINEA PIGS SENSITIZED THROUGH TEETH. *David Weisberger*, B.S. 719

STERILIZATION. AN IMPROVED TECHNIQUE FOR OPERATIONS IN ORAL SURGERY. *Sterling V. Mead*, D.D.S. 727

BIOCHEMICAL STUDIES OF STREPTOCOCCI. VI. RELATION OF OSMOTIC PRESSURE TO AVAILABILITY OF SYNTHETIC MEDIA. *Frances Krasnow*, Ph.D., and *Miriam Reiner*, A.B. 733

WILLIAM J. GIES FELLOWSHIP IN BIOLOGICAL CHEMISTRY AT COLUMBIA UNIVERSITY. *Edgar G. Miller, Jr.*, Ph.D., *Chairman of the Fellowship Committee of the Department of Biological Chemistry* 741

FURTHER COMMENT ON A REFERENCE TO THE CARNEGIE FOUNDATION'S STUDY OF DENTAL EDUCATION. *William J. Gies*, Ph.D. 745

SUPPLEMENT:

I. First District Dental Society of the State of New York. *John Oppie McCall*, A.B., D.D.S., F.A.A.P., F.A.C.D., *Editor* 749

II. New York Academy of Dentistry. Notes from the minutes of the proceedings in 1928–29. *Malcolm W. Carr*, D.D.S., *Editor* 751

III. International Association for Dental Research. Memoranda on the proceedings in 1928–29. *William J. Gies*, F.A.C.D., *Secretary* 756

IV. Quotations from comment relating to the status of dentistry 759

V. Important books recently received by the *Journal of Dental Research* 763

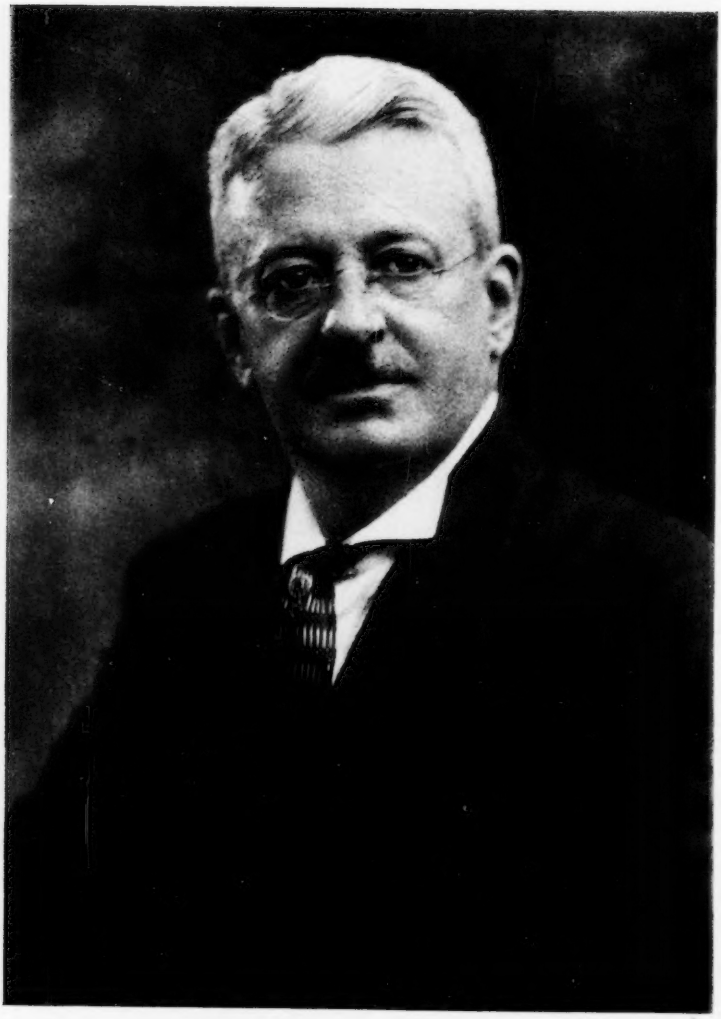
INDEX 767

GENERAL MATTER FOR VOLUME IX:

Announcements iii

In memoriam iv

Contents of the Volume v



FREDERIC AUSTIN PEESO

UNIV. OF MICH.

FREDERIC AUSTIN PEESO[1]

ARTHUR N. CROSS

Frederic Austin Peeso, son of Francis and Hester Peeso, was born in Roseboom, Otsego County, New York, June 25, 1858. He died in Mount Vernon, New York, March 13, 1928, in his seventieth year.

Dr. Peeso received his early education in the public schools of Cobleskill, New York, and then engaged in the jeweler's trade. The mechanical exactness of this work contributed to the skill in mechanics and to the artistic conception that were later to make him an outstanding figure in the practice of dentistry. He was married to Miss Jennie R. Priest in 1880.

In 1887 Dr. Peeso served as an apprentice to Dr. Curtis of Syracuse, New York, and then definitely decided to enter the profession of dentistry. He matriculated at the University of Pennsylvania and was graduated from that institution in 1889. Following his graduation he established himself in practice in Philadelphia, devoting a part of his time to the teaching of crown and bridge work at the University of Pennsylvania Dental School. In 1901 he opened a post-graduate school in Philadelphia, for instruction in crown and bridge work and removable bridge work, at the same time retaining his connection with the University of Pennsylvania. The post-graduate school was conducted successfully until 1910, when it was merged with the University. In the same year Dr. Peeso removed his practice to New York City, remaining there until his death. Soon after coming to New York Dr. Peeso became a member of the faculty of the Dental School of Columbia University as Professor of Crown and Bridge Work.

During a long and active life Dr. Peeso won international renown in his chosen field, and was considered an authority in the subject of crown and bridge work. He devised the split-pin-and-tube attachment for dental prosthesis, and perfected methods for its use in connection with inlays before the day of the casting process, when inlay construction required the burnishing of a fine gold matrix that was

[1] Dr. Peeso was one of the founders of the International Association for Dental Research, in 1920.—[*Ed.*]

filled with a lower-fusing gold. Always an investigator, Dr. Peeso originated a great number of instruments and formulae, many of which are still in use by the profession. In the field of letters he was the author of a text book on crown and bridge work, which was adopted generally in dental teaching. He contributed many chapters in other text books and also wrote many articles.

Dr. Peeso, through his diversity of talents, without doubt did more to advance the practice of crown and bridge work than any other one man, and was the first to develop removable bridge work. His skill, integrity, and erudition commanded the admiration and respect of the dental profession throughout the world, and his delightful personality endeared him to his closer associates. He was a member of a number of dental societies and of the Delta Sigma Delta Fraternity.

Surviving him are his wife, three sons—Frederic E., a mining engineer of Butte, Montana; Frank W., and James B., both dentists practising in New York City—and a daughter, Mrs. Jay F. Barth of Mt. Vernon, N. Y.

A CRITICAL ANALYSIS OF THE DENTAL LITERATURE DEALING WITH THE EFFECTS OF DIETARY VARIATIONS UPON THE STRUCTURE OF TEETH[1]

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An analysis of the dental literature dealing with the observed effects of dietary variations and deficiencies upon the structure and composition of teeth, instituted to obtain a clearer conception of the work already accomplished and its quality, revealed much interesting data. Each paper was carefully read and then grouped as to its scientific worth. We have arranged the papers into three groups. In the first are placed those papers which on the basis of well controlled experiments derive certain well-founded and cautious conclusions. This group has been designated Group A. The second is made up of those articles which, containing excellent reviews and the correlation of research accomplished by workers whose papers fall in Group A, has been designated Group B. The third group includes those papers that do not present any or at most very poor experimental evidence for conclusions stated, which conclusions are based upon poorly defined information. This includes editorials, letters and essays, all of little scientific value. Also in group C, are included papers and reviews that are repetitions, by authors in a number of journals, of work already published elsewhere; and in addition, this last group includes a few clinical papers whose conclusions are not wholly warranted by the amount of evidence presented.

The papers have been judged with this point in mind: Does the article contribute new, well-founded, cautiously interpreted facts, well supported by experimental evidence? In *fig. 1*, papers that measure

[1] Supported by grants from the American Dental Association, the United States Department of Agriculture, Division of Animal Husbandry, and the Dental Staff of the Johns Hopkins Hospital.

up to this standard and the more excellent reviews, are Group A-B papers. Those papers which do not measure up to the above standard

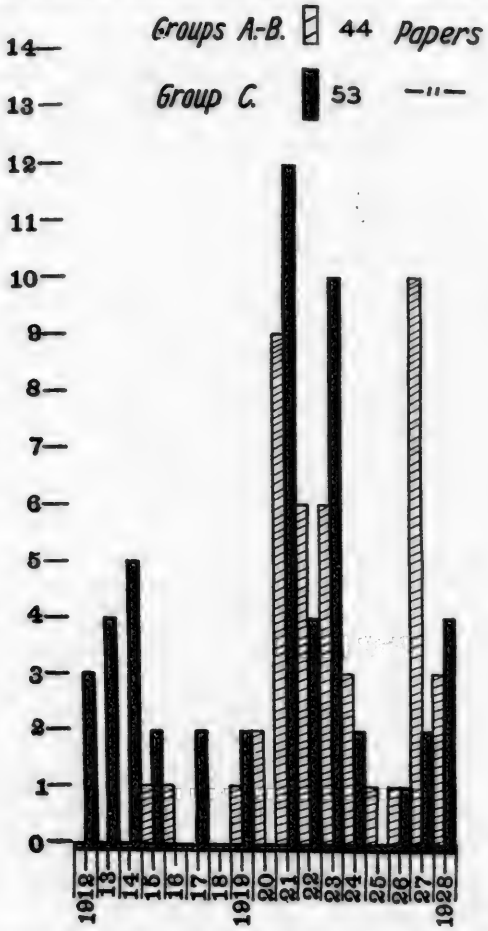

FIG. 1. COMPARISON OF THE NUMBER OF GOOD PAPERS WITH THE NUMBER OF POOR PAPERS PUBLISHED EACH YEAR FROM 1912 TO DATE

Ordinate represents number of papers published. Abscissa represents years

are Group C papers. We have found that the number of papers in Group C is in excess of the number in Groups A and B together.

The comparison of the number of papers in Groups A and B with the number in Group C covering a period of sixteen years, from 1912 to date, is shown in *fig. 1.*

SUMMARY

An analysis of the dental literature dealing with the effects of dietary variations upon the structure of teeth shows that the number of poor papers is in excess of the number of really scientific papers. The analysis covers a period of time from 1912 to 1928, inclusive.

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THE RELATION OF NUTRITION TO GENERAL IMMUNITY[1]

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There are only five essentials in nutrition: proteins, carbohydrates, fats, "accessory" elements, and mineral salts.[2] From the standpoint of physiological chemistry, it makes no difference from what source a protein food is derived, whether animal or vegetable. When it is taken into the body, it is changed in the process of digestion into one fundamental group of compounds called amino-acids. From the amino-acids, the epithelial cells of the intestine synthesize two proteins, serum albumin and serum globulin, which are absorbed into the blood. The serum albumin and serum globulin circulate in the tissues, and through the activity of the different cells of the body they are elaborated into many proteins, which in chemical composition are wholly unlike the stock protein. It makes no difference whether the original amino-acid came from plant or animal protein. When a carbohydrate is taken into the body, it is changed in the alimentary canal into glucose or blood sugar. Then the elaborating cells of the body build from it a number of different substances. What is true of proteins and carbohydrates is also true of the fats. They are brought to the same point. Therefore, a person eating animal food eats a food that is perhaps more nearly representative of his own body than one drawn from vegetables, but he can derive all the necessary nutrition solely from a vegetable tissue.

The types of food essential to normal nutrition are, therefore, comparatively simple. The difficult phase of nutrition is the part that certain directing influences play in the elaboration of food materials

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[2] Water and oxygen are presumably ignored by the author because they are not solid nutrients.—[*Ed.*]

after absorption into the great complexes which keep the life processes going and constitute the normal individual.

The hormones from three ductless glands exercise a great influence on nutrition. The hormone from the *thyroid* directs the growth and differentiation of specialized tissues during intra-uterine life, and in some respects the metabolism of protein in adulthood. We find this illustrated in the cretin, who is born with a congenitally deficient thyroid. In such a child the brain does not develop fully, the skin is coarse, the teeth are poorly developed, and the ability of the tissues to mobilize energy is much below normal. If after thirty years the thyroid becomes deficient, the metabolism of the proteins is reduced from 35 to 55 per cent of normal. Such a condition is known as myxedema. A person who suffers from myxedema cannot meet emergencies. He is abnormal in various ways. His hair drops out, his skin becomes coarse and rough, and his teeth decay early, particularly the molars. Hence, the nutrition of the teeth is to some extent under the direction of this gland.

On the sides of the thyroid gland are four small glands. For a long time they were mistaken for lumps of fat, and a great deal of physiological experimentation was carried on, and misinterpreted, because it was not known that the functions of these glands are entirely different from those of the thyroid gland. These *parathyroids* are important factors in the metabolism of lime. For instance, if the parathyroids are excised from an animal, the wound heals very readily but in five days the animal passes into a state of tetany and dies in that condition. The tetany is the result of a rapid depletion in the blood calcium. Before the removal of the parathyroids, the blood calcium amounted to 10 milligrams per 100 cc. of serum; four days after their removal, the blood calcium had decreased to 1.9 milligrams per 100 cc. When the parathyroids are functionally insufficient, lime *fixation* does not take place and no amount of dietetic manipulation will remedy this deficiency. Tuberculosis is associated with lime deficiencies. When a soluble calcium salt (calcium chloride) is injected into the tissues, it gives temporary relief. The injected lime is quickly eliminated because the parathyroids of such patients are usually incapable of fixing lime. When parathyroid function is adequate, the lime is built into a complex molecule of high molecular weight in the tissues just as the

liver fixes blood sugar as glycogen. The enzymes in the liver bring about a chemical transformation and build the blood sugar into a high molecular compound, which is not diffusible and remains in the body for future purposes. The parathyroids also have the function of detoxication. When they are removed from an animal, it not only loses ability to fix lime in the tissues, but there is abnormal metabolism of protein substances. Instead of deaminizing them into harmless substances, the process stops midway and there are thrown into the blood certain amino compounds that are toxic to the nervous system. Hence the parathyroids have the functions of fixing lime in the tissues and of detoxicating abnormal albuminous products.

Lime is of tremendous importance in the physiological economy of a normal individual. We are accustomed to think of it as a builder of bone, and it is, but it has a more far reaching influence than that—it maintains the normal irritability of the nerve tissues. A muscle nerve preparation with a normal nerve conductivity will, if moistened with a solution that precipitates lime, develop a nerve irritability much above normal. Extreme nervousness observed in some children may be due to lime deficiency either in the food or from faulty assimilation. If for any cause the blood calcium fluctuates, one of two things must happen; either the individual develops nervous irritability or nature reduces the concentration of the other inorganic substances, in which case there is a deep-seated nutritional instability. Lime not only maintains the normal function of the nervous trunks and builds bone, but is also necessary for the normal functioning of the blood corpuscle, and the synthesis of nucleic acid. Therefore many of the problems of nutrition center around the successful assimilation and metabolism of lime. The conditions for its assimilation are most exacting. In the first place an adequate amount of lime must be ingested. In the American diet, the probabilities are that this is often not the case for a number of reasons. First, we do not know how to group our foods. Second, the process of refining by the manufacturer to meet our tastes removes a great deal of the lime. Third, the growing of normal foods on poor soil, where the plants themselves suffer from malnutrition. This point is illustrated in the growing of alfalfa in rich and poor soil. The seeds in both soils develop full-grown plants, but the lime content of the plant grown in poor soil is much below that of the one grown in

rich soil. To keep one's daily physiological processes normal, one must take into the body every day at least fifteen grains of lime. Between the ages of four and fourteen, one must ingest at least three times that much. In the highly refined foods, lime is not present. In the foods grown on very poor soil, lime is not very abundant. Therefore in American life, one of the great problems is to secure in the foods an adequate amount of lime for the hard tissues as well as the soft.

Another great factor that is necessary for lime assimilation is adequate amounts of *direct sunlight.* Dr. Bovey of Harvard conducted an interesting experiment on the relation of sunlight to nutrition. He placed six chicks in a bin and fed them on a balanced ration. He placed six other chicks in another bin and fed them on exactly the same kind of food. In one case the bin was placed in a greenhouse, so that there was plenty of light, *but it had to filter through glass.* In the other case, the bin with the chicks was put out in the sun thirty minutes each day. At the end of the growth period, the chicks not exposed to direct sunlight had diseased parathyroids, rickets, and did not develop sexually. Two of them laid a few eggs, but when the eggs hatched the chicks were so weak they could not peck their way through the shell. That was due to the absence of the stimulating effects of the short wave lengths in the sunlight, which will not pass through window glass. The other chicks developed normally, did not have diseased parathyroids and did not have rickets. They laid the average number of eggs with the average degree of fertility. All this was brought about through the stimulating short wave-lengths of the light. It had to do with metabolism of lime. A child that is living in a semi-darkened room, with no playground, has no prospect of combating tuberculosis. The lime concentration is bound to fall. Stimulating short wave-lengths are absolutely essential. The explanation of that is comparatively simple. Our skin is not only a protective organism, but also in some respects a chemical plant. It is the temporary storehouse of excess sugar but also in the skin there are several substances, one of which is cholesterol, and several other chemical substances. Ultraviolet rays bring about a remarkable transformation in the skin, so that when skin is so exposed and fed to an animal it will cure rickets. These short wave lengths bring about a change in chemical substances in the skin so that it becomes anti-rachitic. In other words it stimulates the metabolism of lime.

Another factor that is very important for the successful assimilation of lime, is a certain degree of *composure*. A person worried by business cares and other causes, no matter what the other conditions may be, will not successfully metabolize lime. An interesting experiment was performed in the University of Wisconsin. A number of cows were put in the stalls, fed a balanced ration, and carefully tested until the investigators were perfectly sure that they were storing lime; that they were taking in more lime than was eliminated. They were in positive balance. This was continued under the same conditions excepting that one of the attendants was given a novel to read and placed where all the cows could see him. That was a new experience for cows. They all went into negative calcium balance. If that is true of a cow, what must be true of human beings?

In order to fix lime in the body, there must be present adequate amounts of two of the *vitamins*. A vitamin is a substance of unknown chemical composition, which may be responsible for the building of enzymes in the body. They are the agents that carry on nutritive processes. Therefore in the absence of vitamins, chemical processes do not occur normally, and in consequence deficiency diseases are developed. *Vitamin A*, fat soluble, is essential for nutrition, especially of the eyes, sinuses, nares, and upper part of the lungs. An animal that is deprived for a sufficient length of time of an adequate amount of this vitamin suffers very great nutritional disturbances. One of the most patent of these is xerophthalmia. After five weeks the lachrymal glands become hardened. The normal epithelium is transformed into a keratin. The eyelids become dry, infected, and later there develops a corneal abscess and the eye explodes. Hence, the resistance of the tissues of the sinuses, nares, and other parts of the body, depends upon an adequate amount of vitamin A in the food. In the absence of it, we are liable to a great variety of infections. In dieting animals to induce xerophthalmia, we have frequently found that the animals die as the result of some infectious disease of the nose or bronchi before eye conditions develop. Sometimes, when animals are under this nutritional deficiency for five weeks, such a flow of mucous from the bronchi develops that it strangles them. In other words, the deficiency of vitamin A leads to marked susceptibility to infection.

The absence of an adequate amount of vitamin A not only brings

about the hardening of the tissues of the eye, but also of other tissues of the body. One of the commonest manifestations is the development of abscesses under the tongue. We had an interesting case in Milwaukee of a young Italian woman in whom every tooth was abscessed and there were also abscesses under the tongue. The condition was caused by nutritional deficiency in vitamin A. The morphological changes that are responsible for the formation of the abscesses are first observed in the epithelium. Some of the epithelial cells become abnormal (they arrange themselves in circles), then they proliferate and produce hard nodules, the epithelium above desquamates and there is formed a hardened mass that ultimately becomes infected—hence the formation of an abscess. Vitamin A deficiency has an influence on the sex glands. Dr. Howe has produced epithelioma of the ovaries at will by depriving guinea pigs of an adequate amount of vitamin A. What is true of the ovaries is certainly true of the tissues of the mouth, kidneys, liver, etc. We may take only enough of vitamin A to be in a state of nutritional instability. Consequently these changes may be taking place in our bodies insidiously, but if they persist long enough, they pass into the chronic stage and we consult a physician, and he diagnoses it as a certain type of disease.

What are the sources of vitamin A? When the chlorophyl in a plant leaf functions in the presence of sunlight, it elaborates this vitamin, which remains in the leaf, but will not stand a great deal of drying nor a great deal of heat. When a seed is formed, some of the vitamin in the leaf passes into the germ of the seed. It does not pass into the endosperm. The American nation is a great consumer of wheat and other cereals. Vitamin A in wheat is in the *germ*. The miller takes out the germ, removes the bran and tissues which might possibly have some of this vitamin in them, and gives us the endospores, which are 77 per cent starch. Therefore the only source of vitamin A during the winter months is green vegetables, such as lettuce, spinach, celery, also cow butter, cod liver oil, etc. A vegetable having a fleshy structure is not good from the standpoint of vitamins. Vitamin A is not very plentifully distributed in vegetables. The best source of vitamin A is good yellow summer butter. When the cow is taken off pasture, and if she is fed exclusively on dry feed, she ceases to store this vitamin in her body. She has the capacity for storing a two months' supply.

She draws on this excess and her milk is apparently normal for about that period. We are able to store in our bodies about a six weeks supply. In all that is three and a half months. By that time we are in the middle of February, the time of colds and contagious infections in general, and it seems as though nature is conspiring against us from that standpoint. That cow milk is deficient in vitamins during winter months is shown by the fact that thirty-day old rats, when fed exclusively on winter milk, did not thrive. Summer milk from the same herd induced normal growth.

Fats or oils in the liver are rich in vitamin A. The one of most importance is the oil from the cod fish. Beginning with the month of February, our natural defense against infections of the nares and bronchi will be minimized by the use daily of cod-liver oil. In one of the schools in New Albany, Indiana, there was an average loss of 3555 hours by 2155 school children because of colds. When they were given cod-liver oil regularly after November first, the loss in school hours was less than 1 per cent. I believe very strongly that the defense of the tissues against infection is dependent on the ingestion of enough of vitamin A. McCollum, one of the leading authorities in the country, says we are not adequately nourished from this standpoint unless we make five per cent of each meal good cow butter.

Three years ago, Mrs. Davis and I did some work for the Indiana State Dental Society. We toured the state in an automobile. Whenever we came to a town where the highways crossed and there was a drug store adjacent, I went in and asked this question: "Aside from your soda fountain stocks, of what article do you sell most to tourists?" In almost every instance the reply was "aspirin." In most dining cars you may see a card advertising that they serve aspirin. People are buying aspirin because of headache, eye strain, or other reasons, but many are buying it because they are in a state of nutritional instability from the lack of *vitamin B*. In the absence of an adequate amount of vitamin B, there are not only nerve disturbances but very profound morphological changes take place in some tissues. Enzymes, which are so essential for carrying on the great complex chemical processes, do not liquefy and do not pass into the circulation. In some cells of the pancreas are large numbers of granules which are the mother substance of one of these enzymes so essential for the digestion

of proteins in the small intestine. When you deprive an animal of an adequate amount of this vitamin, the granules do not liquefy, hence do not develop into true enzymes and pass into the intestine. If one ingests an adequate amount of the vitamin B, the granules liquefy and digestion proceeds normally.

It has been found, in testing the tissues of pigeons, that the normal chemical processes are reduced 50 per cent in the absence of adequate amounts of vitamin B. Glucose injected under the skin is not oxidized. Findley and others have pointed out that enzymes have not formed in the absence of this vitamin and therefore the normal chemical processes do not occur. The nuclei of the body cells are the chief agents in most chemical processes in the body. In the persistent absence of vitamin B, the production of nucleic acid is greatly reduced. Hence, there is great disturbance in the metabolism of the nucleic acid group. McCarrison produced, through deficiency of this vitamin in lower animals, diseases which are comparable with certain human diseases. There may develop lesions in the epithelium of the small intestine. The absorbing villi of the small intestine become atrophied and lose their motility, and hence normal absorption in the small intestine is greatly reduced.

In the absence of this vitamin, there is not only lack of absorption in the small intestine, but also change in the germinal epithelium. A male bird fed short for one hundred and fifty days, from the standpoint of vitamin B, becomes sexually impotent. If the female bird is fed in like manner, ovulation ceases and sex is dead, but she has power of recovery. Normally the blood possesses the capacity of neutralizing 40 to 50 volumes of carbon dioxide for each volume of blood. In protracted absence of the vitamin, there is lowering of the alkali reserve. The result is that there is developed a mild state of acidosis, or tendency thereto. Such studies suggest that when one is nutritionally unstable from the standpoint of vitamin B, the chemical processes of the body are reduced below normal, and one is open to all sorts of susceptibilities to contagion.

Vitamin C is water soluble and normally present in fruit juices, if the fruit is grown in abundant sunshine. The absence of vitamin C produces profound histological changes in the body. In normal bone the osteoblasts are arranged alongside of the bone very much like the

palings on a fence. The odontoblasts in the dental pulp are arranged in the same orderly manner. In the absence of vitamin C, the osteoblasts migrate into the soft tissues or they become elongated, and look like fibroblasts or connective tissue cells. The result is that some of the osteoblasts migrate into the soft tissue and there produce bone tissue. In the absence of an adequate amount of vitamin C, there is failure to form cement substance, which is the basis of the repair of blood vessels and the walls become thin. There is then a tendency to hemorrhage. The bone marrow becomes almost incapable of normal functioning in the absence of an adequate amount of this vitamin. When animals are deprived of vitamin C for a long time, the dental pulp and dentine are changed.

In depriving an animal of *vitamin D* for a long time, in the early stages of its growth, the enamel of the teeth is changed. Without vitamin D, neither dentine nor enamel of good quality is formed.

The distribution of vitamins in foods is fairly well defined. Vitamin A is present in cow butter and in fish oils. There is vitamin B in the working part of any plant. Vitamin C is especially abundant in fruit juices. Vitamin D is most abundant in butter, cod liver oil, and sparingly in the green tissues of plants.

All foods can be divided into two groups: storage foods and biologically active foods. All the cereal grains are storage foods and not one is adequate for nutrition. An animal of any breed, even a rat, if fed wholly on cereal grain, will die prematurely. The blending of grains is not adequate for growth and well being. Beans, tubers and muscle meats are also storage foods. In the foods from biologically active tissues, the protoplasm is of a high order and contains the vitamins. Until 1840, in Greenland and Iceland, there were no such words in the language as caries and pyorrhea. Amundsen, or another of the Arctic explorers, exhumed skeletons that had been buried for three to five centuries and did not find any sign of caries. In 1840 American groceries were introduced into Iceland, and now the natives have those diseases. Not that it is the fault of the grocer. It lies in the grouping of our foods. The things that the grocery store furnishes in largest amounts are generally storage foods—cereals, legumes and muscle meats. We are feeding on that type of food, and we have dental caries and other nutritional disturbances. A good many years

ago they could not rear lions in captivity. They died of a pulmonary infection. An Englishman watched a native lion eat. He tore open the carcass of his kill, ate the soft bones of the ribs, and the liver, pancreas, and spleen. He then licked out the brain and rolled the carcass down the hill for the jackals to finish. He had discarded the storage parts of the body and eaten the biologically active, where the vitamins are stored.

How does it happen that the people of Greenland and Iceland can live when they do not have vegetables and fruits? The native eats muscle meats only in times of stress. He drinks the blood, cracks the bones and eats the marrow, the intestines and the oils, and in so doing he fulfills all the necessary conditions for a normal nutrition. The great shortcoming in American diets is the tendency to eat storage foods at the expense of the more biologically active foods. Our dietary habits are reflected in the diseases of the hard and soft tissues of the mouth.

As far as we can observe, there are two types of physical condition that accompany pyorrhea, depending upon whether the pyorrhea is of long or short duration. In the case of *acute* pyorrhea the blood calcium is generally much higher than normal, and the blood sugar is also above normal. The body establishes its best defense against streptococcic infection when it develops ionic calcium. Usually in acute pyorrhea there is a low alkali index. The body establishes this defense against streptococcic infection by drawing on the alkali reserve. The theory is that high ionic calcium is maintained by absorbing the alveolar process. In cases of *chronic* pyorrhea the opposite is true. We find low ionic calcium and a low alkali reserve. The causes which lead to this condition are obscure. Experimental evidence indicates that there are at least two factors; namely, absorption of toxins from pus pockets, and nutritional deficiency in vitamin B, because in those animals starved of vitamin B there is a drop in the alkali reserve.

DISCUSSION

Arthur F. Coca, M.D. (New York City): I must say at once that I know very little about nutrition. I must confess, also, that I do not know much about immunity, although I have spent most of my personal life in the study of immunology. I was so fully absorbed in this most interesting talk that

I almost forgot that I had to follow. I caught myself just in time to take a few notes on Dr. Davis' remarks as far as they concerned immunity. It seems that he expected me to correct the possible false impression that immunity is *all-dependent* upon nutrition. That, of course, Dr. Davis knows is not the case. He has shown that nutritional deficiencies of various kinds can put the body into such a state that it is less than normally resistant to infections. On the other hand, most people that are properly fed on what even Dr. Davis himself has referred to as a balanced diet, are immune to infections while some of them are not.

Before the modern teaching on nutrition was begun, all of us knew the importance of nutrition in the cure of tuberculosis, that is, the ultimate establishment of an immunity. Yet, we know that 95 per cent of the other people, some of whom are not at all in a good state of nutrition, have passed through a tuberculous infection at some time in their lives. It is a well-known pathological finding that about 95 per cent of the population have passed through a tuberculous infection and have overcome it, but many of these people have been in a low state of nutrition. Certainly more than nutritional deficiency is necessary for the presence of a critical susceptibility to tuberculosis that runs a progressive course.

As to the infections of the cornea, and abscesses of the tongue, that followed the deficiency of vitamin A, these again are special cases. They do not explain the occurrence of abscesses in other parts of the body or the usual occurrence of abscesses, which are certainly not all due to nutritional deficiency.

Regarding the Febraury zone of colds, I have to express quite a little doubt as to the existence of such a limited "cold zone." In my own experience, I have from two to four colds a year. They may occur at any time between September and April or May, usually within the period of those months and very rarely in the summer time. Why are colds more frequent in the winter than in the summer? This need not be because of any nutritional deficiency. It can be explained on the basis of the fine experiments of Teague and Strong explaining the epidemic occurrence of pneumonic plague. They found that bacteria sprayed into a warm dry air died in a minute and a half. When the same conditions were duplicated as to space, but with a cold and damp atmosphere, the same bacteria remained suspended and alive in that air for two or three hours. That makes much difference as to whether we are going to pick up a cold from a man's sneeze in the subway. If the air is warm and dry, we have a very good chance of coming there just a minute and a half too late to catch it, whereas if it is cold and damp, the chances are against us.

Immunity to bacterial disease is exhibited to two great classes of bacteria: those that produce toxins and those that do not. We understand perfectly the immunity to the bacterial intoxications. That is almost always dependent upon antitoxin, which may be present naturally in the blood without our having passed through an infection. Take, for example, diphtheria; a child may be born immune to diphtheria and never become susceptible to it, and in every one of these cases antitoxin naturally in the blood explains the immunity. In such cases, of course, nutrition could have no bearing; we know of no instance where the antitoxin production has been affected by a nutritional deficiency.

An interesting instance, which covers both of these classes, is that of the streptococcus of scarlet fever. This organism produces a toxin, and it is the toxin that causes the acute symptoms of scarlet fever. But the organism has also invasive properties and, after the acute intoxication has been overcome by the development of antitoxin, the organism sometimes displays its invasive properties and produces the well known abscesses, especially in the mastoid and the middle ear, as well as other parts of the body. Now in this case, a person may be immune to the toxin, but susceptible to the invasive microörganism. Even those of us who never have had scarlet fever, which is merely an expression of the intoxicating property of these bacteria, may have abscesses induced by these bacteria without any symptoms of scarlet fever whatever. In other words, organisms that are indistinguishable from the scarlet fever streptococcus and which produce the toxin of the scarlet fever streptococcus have been isolated out of abscesses in individuals that had never had any symptoms of scarlet fever.

We do not know the mechanism of immunity to the infections. I can say we practically know nothing about this. These bacteria, when they are injected into animals or when they are injected into human beings, stimulate the production of various anti-bodies, but none of these anti-bodies has been identified as the mechanism of the immunity. And so we can say we really know nothing about it. Therefore, we are unable to study in the human being the effects of nutrition upon the establishment of immunity to the infectious diseases.

Now after having made these rather critical remarks, I should like to return to what Dr. Davis told us of the observed and known effects of nutritional deficiencies upon immunity. These observations still exist just as he has described them. They are perfectly correct, but they do not explain the whole of the development or regulation of immunity to infections in individuals that are not suffering from nutritional deficiency.

President Arthur H. Merritt: I am sure some would like to ask questions of Dr. Davis or Dr. Coca. I think we would all be glad if Dr. Davis, in summing up his talk, would outline very briefly the common every-day foods in which each of the vitamins may be found so that we may better understand in our work where the vitamins needed in an individual case may be obtained.

In Chicago, last week, Dr. Davis gave a very interesting talk on pyorrhea alveolaris. The impression was rather general that in his belief the real factor in all pyorrhetic infections was nutrition. When the subject was open for discussion, one of the members present asked why it was that many of the periodontists, who knew nothing about nutrition and did not consider it at all in the treatment of cases, got excellent results in many instances, seemingly proving that there must be other factors than the group outlined by Dr. Davis. I think those of us engaged in that work quite agree that this is true.

Leuman M. Waugh, D.D.S. (New York City): Dr. Davis tells us vitamin A is found in *yellow* cow butter. If this butter, made from summer milk, will not store vitamin A so that it is available during the months when milk is deficient in vitamin A, what is a substitute?

I should like to comment, if I may, upon my personal observations of people in the north. I have not studied Greenland or Iceland, but I have had the privilege of studying the Labrador Eskimo, and have visited the nomadic tribes which have not come into contact with the white man. In speaking of the form of the teeth of northern races, Dr. Davis seems to have the impression that there are very few developmental defects on the occlusal surfaces. We have been told always that the Eskimo, although a race of small stature, has the largest and strongest jaws of any in the world and that also the incidence of caries is less in the Eskimo. This is borne out by the collection of skulls in the American Museum of Natural History. In going among the Eskimos one finds a peculair condition, which only partly bears out what Dr. Davis has said in this regard. A difference is noted in different locations. In the southern part of Labrador, three hundred miles north of Battle Harbor, where the Eskimo can get molasses, sugar, gum drops, cheap candy, and white flour, the teeth are in deplorable condition. I have never seen mouths in worse condition. It stimulated my desire to go farther north. The farther north one goes the more tribes one meets, for every hundred miles one finds a new tribe. Here the only means of travel is by way of the dug-outs. They never travel distances in winter. As you go inland you find they are very much isolated, and intermarriage between brothers and sisters is common.

In the northern races we find an entirely different type of Eskimo. He has no carbohydrate food at all. Out of seventy-eight mouths I examined, I found twenty-six cavities of decay. I found very few mouths with thirty-two teeth all in excellent occlusion. They were worn down, and there were no evidences of grooves or fissures. The grooves wear out because of the coarseness of the food which they eat—in the women especially, because they chew the frozen boots of their husbands, sometimes working two or three hours until they become soft as chamois. In young women of seventeen years of age the occlusal topography is destroyed from abrasion due to chewing the boots and raw food. In many of the men, at thirty years of age, half of the occlusal line is worn down. Their teeth are flint-like in hardness, and have hard, brown, polished surfaces. In many women at twenty-four to thirty the teeth are worn to the gum line from chewing the coarse food. I think the absence of grooves and pits in the occlusal surface of the teeth is due to this abrasion.

In this section of the north, where the Eskimos eat an almost entirely protein diet, such as dry seal meat, dried fish, seal blubber (which is fatty), and things of that sort, they have deep heavy deposits on their teeth. A periodontist should go up there and study the conditions. In two half days I extracted perhaps thirty teeth in addition to the examinations and impressions that I made. The loss of these teeth was caused through trauma, breaking of the teeth, accident, or through the heavy deposits. The teeth were dark greenish-brown. I did not have my scalpels with me, but I tried scaling the teeth to determine how dense the deposits were, and found them just like flint. Destruction of the supporting structures of the teeth, by the encrusting deposits, causes loosening of the teeth, and destruction of the alveolar process and periodontal membrane. Speaking of pyorrhea: I do not believe that pyorrhea is primarily produced by bacteria. It is a secondary effect of chronic hyperemia. The resistance of the tissue is lowered sufficiently to enable bacteria to live there. They are found in abundance around the gum margin because here there is a free trench. I found practically no pus in these cases. I could observe it only by pressing with my finger tips from the high part of the gum down to the margin. There was not that flow of pus we ordinarily see in many of our cases here.

I think the lack of dental caries in the Iceland and Greenland and northern Labrador Eskimo is due to the fact that the natives have not had carbohydrate food. Fatty foods do not decompose in the mouth. The only foods from which acids can be formed are carbohydrates and the Eskimo do not have them. They have no butter. The Moravian missionaries have taught them to dip their bread into cod liver oil. The health of the Eskimo

is better than that of the natives of Labrador and the Newfoundlanders on the West Coast. When white man's food is taken to the Eskimo, his teeth decay.

I think we must be a little careful when we say there are no grooves and fissures in the Eskimo teeth, and no developmental defects. I think these are obliterated through wear. In addition to the absence of carbohydrate foods, the natural wear of the teeth is one of the important factors in their freedom from dental caries.

Dr. Davis: I did not mean to make the statement that there were no fissures. The skulls to which I referred were taken from people who lived close to us in Arizona, not from the far north. I obtained these skulls because I was also able to ascertain the history of the people themselves. In the particular skull to which I referred there were no fissures. What I mean by a pit or fissure is a place where the enamel has not coalesced, where it would be easy for whatever it is that causes decay to make its inroads.

As far as pyorrhea is concerned, I do not know what causes it. I believe susceptibility to it is encouraged by the use of the storage foods so likely to come in cans.

Vitamin A is quite resistant to temperatures and quite susceptible to oxidation. If a product is made and then enclosed so that there will be no oxidation, it ought to keep for from two to five years and be perfectly reliable. We have found that we can keep cod liver oil for at least two years without deterioration, if we prevent air from affecting it. I have no doubt that would be true of cheese also.

In connection with my reference to *yellow* butter, it is an interesting thing that vitamin A is closely associated with the yellow pigment.

All the cereals and white flour, including all of the tubers and legumes in the unsprouted stage, are classed as storage foods. They yield energy but do not produce the conditions for the other necessary chemical processes in nutrition. The accessory foods, including the vitamins, are the ones that bring about the conditions for the normal chemical processes. The foods that contain vitamin A are thin green leaves, and the sperm of cereals. The only source in which there is a practical, adequate amount in winter is butter, carrots, and cod liver oil. Vitamin B is widely distributed in the coating of cereals. It is also in the working parts of plants, celery (if it is green), lettuce, and spinach, which are also rich in lime. Malted bran is an excellent food from that standpoint. Vitamin C is most richly distributed in raw fruits and vegetables. Vitamin C will stand neither drying, storage, nor heat. In canning tomatoes, if the process of packing and heating in

fifteen minutes is used, a great deal of vitamin C is conserved. Tomatoes are the best canned product because of the method of canning employed. Usually, if a person eats, in two meals, fifteen per cent in raw fruits and vegetables, an adequate amount of these vitamins will be obtained. Vitamin D is not so widely distributed. It withstands heat and also oxidation. Vitamin D occurs sparingly in butter. Cod liver oil contains it in largest proportion.

President Merritt: What do you regard as an anti-neurotic diet?

Dr. Davis: If it has not been caused by focal infection, but is the result of nutritional deficiencies, I would cut out red meats and use white meats and raw vegetables, largely because they are rich in vitamin B, neuritis from a nutritional standpoint being due to that deficiency. Incidentally the vicious effects from deficiency vitamin D are not on the nerves at all.

I would like to say a word in regard to solarized cod liver oil. Our experience has shown that ordinarily in the distribution of the lime in the blood the ratio of the ionized lime, in the form of globulin, is about fifty per cent. If you buy activated or solarized cod liver oil it raises the available ion rapidly. Raw cod liver oil does not do it. Evans and many other investigators have demonstrated pretty clearly that the prolongation of sex life, as it should be for a long time, depends on another vitamin, called vitamin E, which is not widely distributed, but is found sparingly in the leaves of vegetables, and abundantly in the sperm of cereals, particularly wheat and corn. Therefore in making a tasteless cod liver oil, we activate it—92 parts of cod liver oil are mixed with 8 parts of a blend of the oils from these cereals so as to provide vitamin E. Some of the nutritional effects we have had in post-operative anemia have been remarkable, particularly in children. I believe all children should have such treatment.

Leland Barrett, D.D.S. (*New York City*): I am interested in the possibility of securing tasteless cod liver oil and the practical application of it for children. Is it commercially available?

Dr. Davis: You know there are always some seniors who need help. We have a little laboratory under my direction where they make several products for physicians and dentists. One of these is activated cod liver oil. If you like it and it is effective, after you try some samples, I will turn the matter over to you and you can take it up with the boys. Regarding cod-liver-oil tablets, I do not believe in them. You cannot make them im-

pervious and the cod liver oil does not stand oxidation. Expose it to air and it fails.

Joseph D. Eby, D.D.S. (*New York City*): Perhaps you know Professor Park, Professor of Pediatrics at Yale. I understand that, for eighteen or twenty years at Johns Hopkins, he devoted himself to the nutritional study of children. In a paper he read five years ago before the New York Society of Orthodontists, he gave the results of his investigations of various available emulsified, tasteless, and deodorized cod liver oil products. He explained that the cod liver, as it is extracted from the fish at different periods of the year, varies and stated that a cod liver oil manufactured by Patch was not only one of the least objectionable but clinically one of the most effective. I should like to have your opinion of this product.

Dr. Davis: You can't create a vitamin. The only thing you can do is to bring about a condition for the ionization of the lime. The cod liver oil we use has to be certified by a government agent. We pay twelve dollars a barrel for that service. It must measure 1200 units per gram of vitamin A and 380 units of vitamin B. I do not know of any native cod liver oil that ranks higher. In our treatment we require that it shall not be exposed at all to the sunlight or air.

Dr. Eby: Is there any more illuminating book from which we can obtain nutritional information than McCollum's latest book?

Dr. Davis: In my opinion McCollum made one mistake. He put in the whole résumé and then said: "This has all been proved to be fallacious." From the standpoint of the layman, busy at something else, the dietary problem is not very easy to solve. The books that give detailed dietaries only lead to confusion. I tried to show you that the matter of nutrition from the standpoint of foods is comparatively simple. This little leaflet I give you will indicate the distribution of the vitamins. Lime presents the one problem. We reinforce lime in our foods by using a common table salt, to which has been added precipitated calcium phosphate (20 per cent), which acts as a drier. That reinforces the lime and keeps the teeth hard. We hope so at least.

Henry W. Gillett, D.M.D. (*New York City*): Is there any foundation for the claim advanced by certain people that protein and starch digestion cannot go on simultaneously—that we ought not to mix the two in our diets?

Are the new forms of glass that are supposed to be very helpful in giving us the effects of summer sunlight effective for the city dweller?

Dr. Davis: It remains to be seen what the effect of the new glass will be. Constructed to allow the ultraviolet rays to pass through, it should be quite the thing. Some of the hospitals have installed this glass in their sun parlors. The test has been running only two years, but I think it will be perfectly all right.

In the chemical processes in the body, the proteins and starches are independent. One set of enzymes work on one, another on the other. There is no reason why anybody should eat two starches, but I cannot see why they should not eat an albumen and a starch.

Charles F. Bödecker, D.D.S. (New York City): When you examined the patients in the tubercular hospitals, did you not find considerable caries there? I ask that question for the purpose of learning your view, and also because I am trying to determine whether a high calcium content means immunity to dental caries.

Dr. Davis (in conclusion): It depends upon the condition of the alkali reserve. If it is normal and the calcium content is high, there is defense for the tissues of the mouth. If on the other hand the alkali reserve is low and the ionic content high, this is not the case. In the treatment we are working on, we are proceeding along that line. The first thing we do is to raise the alkali reserve so as to protect the tissues high in calcium content. The polymorphonuclears will draw upon this tissue for the lime.

BIOCHEMICAL STUDIES OF STREPTOCOCCI. I. A COMPARATIVE STUDY OF SOME COMMON MEDIA FOR THE CULTIVATION OF STREPTOCOCCI[1]

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CONTENTS

I. Introduction 29
II. Media 29
III. Cultures 30
IV. Procedure 31
V. Storage of cultures 33
VI. Summary 34
VII. References to culture media 35

I. Introduction

This study was undertaken, some years ago, in order to ascertain the differences in the availability of certain media for the growth of streptococci.[2] Since optimum environmental conditions prolong the life of microörganisms, the values of the media were estimated from the degrees of viability of the cultures. The methods employed and data obtained are detailed below.

II. Media

(*a*) *Hemolyzed blood-glucose-veal infusion agar*: 75 grams of Bacto-veal and 500 cc. of tap water were heated in an Arnold sterilizer for two hours, and filtered through filter paper. The hot bouillon easily dissolved additions of 10 grams of Bacto-peptone and 5 grams of sodium chloride. After cooling, 10 cc. of the filtrate were adjusted, with 0.05 M solution of sodium hydroxide, to a hydrogen ion concentration

[1] Krasnow and Gies: Proceedings of the International Association for Dental Research, *Journal of Dental Research*, 1928, viii, p. 439.

[2] A study of root-canal treatment, in collaboration with Drs. M. L. Rhein and Joseph M. Levy, of New York City, made this inquiry desirable.

of 7.9 pH. The calculated amount of M solution of sodium hydroxide was added to the remainder, and the liquid set in an Arnold sterilizer for fifteen minutes. Finally, the broth was mixed with 500 cc. of 3 per cent solution of agar-agar and sterilized in an Arnold apparatus. The medium was tubed as needed—0.1 cc. of sterile hemolyzed defibrinated sheep blood and 0.25 cc. of sterile 12 per cent glucose solution were added to 3 cc. portions, and these allowed to solidify in slants with all of the tubes at a uniform angle.

(*b*) *Same as* (*a*), *except* that 0.1 cc. of sterile 1 per cent glucose-beef infusion broth was added after the medium had solidified.

(*c*) *Rosenow dextrose-brain broth* (*1*)

(*d*) *Jablons liver-peptone medium* (*2*)

(*e*) *Huntoon hormone broth* (*3*)

(*f*) *Huntoon hormone agar* (*3*)

(*g*) *Beef-liver agar*: 500 grams of fresh, hashed beef-liver were infused in 500 cc. of tap water for three hours at 55°C., and the liquid strained through an iron-wire sieve. Then 10 grams of peptone and 5 grams of sodium chloride were added, and the mixture placed in an Arnold sterilizer until solution was complete. The reaction was adjusted to a 7.9 pH. Finally, 500 cc. of 3 per cent agar-agar solution were added, and the liquid sterilized in an autoclave for fifteen minutes at fifteen pounds pressure.

(*h*) *Beef-brain agar: Same as* (*g*), *with brain substituted for liver.*

(*i*) Hastings' modification of the North (4) medium.[3] A mixture of one pound of meat and 1000 cc. of water was boiled for one hour, when the liquid was filtered through an iron-wire sieve and the filtrate made up to 1000 cc. This fluid, after additions of 10 grams of peptone and 5 grams of sodium chloride, was boiled for twenty minutes in an Arnold sterilizer. Then, after the addition and solution of 15 grams of agar-agar and 20 grams of gelatin, the medium was made neutral to phenolphthalein, and sterilized and tubed as in North's procedure.

III. Cultures

All of the cultures were streptococci isolated from infected human teeth.[4] The series contained strains belonging to the three different

[3] Hastings' formula as given to Dr. Rhein and by him presented to us.

[4] The cultures were obtained from Drs. Rhein and Levy.

groups, classified according to the reaction with defibrinated sheep blood, beef infusion agar (0.5 cc. of blood to 9.5 cc. of agar). The strains differed also in age, i.e., in the time that elapsed from isolation to the beginning of the test, this period ranging from several days to several months. Immediately after isolation, each culture was planted in 2 cc. of defibrinated sheep blood, incubated for four hours at 37°C., and stored in an ice chest. Before starting a test, the culture was transplanted to 1 per cent glucose-beef infusion broth and incubated for eighteen hours, thus serving as a seed culture.

TABLE 1

Comparison of amounts of growth, series A

MEDIUM	GROWTH	
	Estimate	Description
Hemolyzed blood-glucose agar + broth (b)	++++	Very abundant on all except one, where it was slight
Rosenow dextrose-brain broth (c)	++++	Very turbid; not comparable with solid media
Jablons liver-peptone broth (d)	++++	Very turbid; not comparable with solid media
Huntoon beef-heart broth (e)	+++	Quite turbid, except one, which was clear
Huntoon beef-heart agar (f)	+++	Quite abundant, except one, which was slight
Liver infusion agar (g)	++++	Very abundant, except one, which was slight
Brain infusion agar (h)	++	Slight
Hastings gelatin agar (i)	++	Slight

IV. Procedure

The test medium was inoculated with one loopful (platinum loop, 4 mm. in diameter) of culture from the seed tube, kept at room temperature (23–25°C.) for sixty hours, and then incubated for twenty-four hours at 37°C. At regular intervals, transplants to glucose-blood agar plates were made, incubated for twenty-four hours at 37°C., and examined for growth. If growth was slight or doubtful, subcultures were made to test the initial results.

Series A. Each culture tube contained 3 cc. of medium. Tests

were made with fifty strains of streptococcus: 3 S. hemolyticus; 19 S. viridans; 28 S. non-hemolyticus. The experiment was continued for twenty-six days; subcultures were made 5, 7, 9, 11, 13, 15, 20 and 26

TABLE 2

Summary of results of series A, showing comparative viability

MEDIUM	NUMBER OF CULTURES THAT DIED IN A SPECIFIED NUMBER OF DAYS AFTER INOCULATION							
	5	7	9	11	13	15	20	26
Blood agar + broth (b)	0	0	0	0	0	0	0	3
Rosenow broth (c)	0	0	0	0	0	0	0	0
Jablons broth (d)	0	0	0	0	0	0	0	0
Huntoon broth (e)	1	1	1	1	2	2	2	2
Huntoon agar (f)	0	0	0	0	1	3	3	3
Liver agar (g)	0	0	0	0	0	0	0	1
Brain agar (h)	0	0	0	0	0	1	1	2
Hastings agar (i)	1	1	1	1	1	1	1	1

TABLE 3

Summary of results of series B, showing comparative viability

MEDIUM	NUMBER OF CULTURES THAT DIED IN A SPECIFIED NUMBER OF WEEKS AFTER INOCULATION				PER CENT VIABLE AFTER TEN WEEKS
	4	6	10		
			Number dead	Number transplanted from dry medium	
Jablons broth (d)	0	0	0	0	100
Rosenow broth (c)	0	1	2	0	96
Liver agar (g)	0	0	5	3	96
Brain agar (h)	0	1	3	1	96
Huntoon broth (e)	0	1	3	0	94
Hastings agar (i)	0	0	3	0	94
Huntoon agar (f)	0	3	4	0	92
Blood agar + broth (b)	0	13	36	0	28

days after inoculation. The amounts of growth are shown in *Table 1*. The results of over 3000 transplants are summarized in *Table 2*.

Series B. Each culture tube contained 6 cc. of medium. Tests were made with fifty strains of streptococcus: 1 S. hemolyticus; 21 S. viridans; 28 S. non-hemolyticus. The experiment was continued

for ten weeks. Transplants were made at intervals of 4, 6, and 10 weeks after inoculation. *Table 3* summarizes the results of 1250 transplants.

Series C represents a comparison between hemolyzed blood-glucose-veal infusion agar (a), and hemolyzed blood-glucose-veal infusion agar to which glucose-beef infusion broth had been added (b). Each culture tube contained 6 cc. of medium. Tests were made on 29 strains of streptococcus: 13 S. viridans, and 16 S. non-hemolyticus. The experiment was continued for ten weeks. Transplants were made

TABLE 4

Comparison of amounts of growth, series C

MEDIUM	GROWTH	
	Estimate	Character
Blood agar + broth (b)	++++	Very abundant
Blood agar (a)	+++	Abundant

TABLE 5

Summary of results of series C, showing comparative viability

MEDIUM	NUMBER OF CULTURES THAT DIED IN A SPECIFIED NUMBER OF WEEKS AFTER INOCULATION			PER CENT VIABLE AFTER TEN WEEKS
	4	6	10	
Blood agar (a)	0	1	5	83
Blood agar + broth (b)	0	7	20	30

4, 6, and 10 weeks after inoculation. The significant differences are indicated in *Tables 4* and *5*.

V. Storage of cultures[5]

The above findings suggested this question: Can streptococci be stored in the Jablons medium (d) for periods longer than ten weeks? To ascertain the answer, on May 21, 1921, twenty-eight strains were transplanted from their blood cultures to the Jablons liver-peptone broth. One loopful of culture was added to 10 cc. of broth, and each

[5] We are indebted to Margaret L. Rosenberg for effectual coöperation in some of the experiments in this section.

mixture incubated for sixteen to eighteen hours at 37°C., and then placed in an ice chest at 10–15°C. At intervals of several months subcultures were made in new samples of the same medium and also on blood agar, with the following results:

DATE OF SUBCULTURE	NUMBER OF STRAINS VIABLE	PERCENTAGE OF STRAINS VIABLE
12/19/21	28	100
5/19/22	28	100
12/19/22	28	100
3/12/23[6]	26[6]	93[6]
6/27/23	28	100
10/11/23	28	100
6/ 6/24	28	100

The age of most of the strains used in the tests in this section was more than a year. Tests were also made with freshly isolated strains of streptococcus (three days old). Although we have had occasion to work with only three cultures of this type, the results were analogous to those above. Moreover, similar data accumulated for other organisms (B. coli, B. proteus, B. neopolitan, B. megatherium, B. subtilis) suggest that a great number of bacteria belonging to very diverse groups may be successfully stored for long periods in the Jablons medium.

VI. Summary

The foregoing data show that these media were not equivalent in availability for the nutritional needs of our strains of streptococcus.

No inhibitive effects were discernible four weeks after inoculation, but differences were apparent in six weeks and very marked in ten weeks.

All of the media are useful for ordinary purposes. But, when longevity of culture without transplant is desired, the Jablons medium is the most useful, for it supports the life of the greatest number of organisms for the longest period.

[6] The two cultures that did not grow when transplanted were contained in tubes that showed practically no fluid over the liver (d). Since the remaining twenty-six tubes were also very low in fluid, 10 cc. of peptone water (d) were added to each of the twenty-eight tubes. All were then incubated for sixteen to eighteen hours, and returned to the ice chest. The next subculture gave the results shown for June 27, 1923.

In the last columns of *Tables 3* and *5* the media are listed in the order of preference for the cultivation of streptococci. The figures in these tables may be interpreted as the efficiency values of the media.

The Jablons broth is an excellent medium for the storage of streptococci. Twenty-eight strains, stored for three years without transplanting, showed undiminished viability.

Collateral tests indicate that the Jablons broth is also useful for the storage of a great number of bacteria belonging to diverse groups.

VII. References to culture media

(1) ROSENOW 1919 *Journal of Dental Research*, i, p. 205.
(2) JABLONS ET PEASE 1918 *Comptes rendus hebdomadaires des séances de la Société de Biologie et des filiales*, lxxxi, p. 1072.
(3) HUNTOON 1918 *Journal of Infectious Diseases*, xxiii, p. 169.
(4) NORTH 1909 *Journal of Medical Research*, xx, p. 359.

THE BACTERIAL DESTRUCTION OF DENTAL ENAMEL

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AND
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The title of this paper appears paradoxical in the light of the accepted theory of dental caries. In our present conception of the destruction of the enamel, Bacillus acidophilus forms lactic acid, which dissolves the enamel and thus initiates the process of dental caries. It is believed that the disintegration of the enamel progresses in the same manner as the dissolution of a lump of marble in a beaker of hydrochloric acid. This, we believe is only half, although from a clinical point of view the more important half, of the truth of the carious destruction of the enamel. Specimens of carious enamel, made by our celloidin decalcifying method, show that it is a more complex process than heretofore considered, one which more closely parallels the destruction of the dentine.

The carious destruction of the dentine is dependent upon the action of two factors, which were well brought out in W. D. Miller's memorable work and designated as the chemico-parasitic theory of dental caries. It appears that the dentine is first decalcified by the action of an acid, and afterward the organic basic substance is destroyed by the proteolytic action of bacteria.

Basing our observations upon a careful study of slightly carious enamel, we believe that the destruction proceeds in the following manner:

(1) The formation of mucigen plaques (first described by J. Leon Williams and by G. V. Black) containing the Bacillus acidophilus (suggested by R. W. Bunting and others), which makes lactic acid.

(2) The lactic acid initiates decalcification of the enamel under the plaque to a slight depth.

(3) Cocci, probably diplococci or streptococci,[1] exert a proteolytic action upon the matrix of the enamel.

The last two processes alternate, and thus there is formed in the enamel a cavity that soon penetrates into the dentine.

We have been able, for the first time, to present specimens showing individual bacteria in the very act of destroying enamel. Even if this investigation had gone no further than to indicate that the destruction of the enamel is accomplished by more than one agency, i.e., by decalcification plus a proteolytic action, we could say that some progress had been made in the solution of the problem of dental caries.[2] The investigation has, however, gone further, and suggests proof of a theory long maintained by J. Leon Williams concerning the embryonic formation of the enamel.

In order to comprehend this matter, it is necessary briefly to review the two theories of the embryological formation of this tissue:

(a) One theory maintains that the enamel-forming cells, the ameloblasts, in growing outward, constantly lengthen and are themselves calcified to form the enamel, each cell forming a single enamel rod. If this theory is correct, then the enamel is a calcified, epithelial *tissue*.

(b) The other theory maintains that the enamel-forming cells, the ameloblasts, secrete the enamel, they themselves remaining intact until their function is fulfilled. If dental enamel is formed in this way, it is a simple *secretion*.

Let us turn now to the new data, brought out by the use of special methods of decalcification, that indicate which of these theories is correct. The protein in enamel has heretofore been regarded by us as keratin, which also occurs in the outermost layer of the skin, covering the entire body with a resistant surface. This layer of horny cells protects the individual against external irritants, particularly against the attack of bacteria, which do not destroy or digest keratin.

The accompanying photomicrographs of carious enamel clearly show

[1] The opinion of Professor Jobling, Pathologist at the College of Physicians and Surgeons of Columbia University, and of Dr. Enright, Bacteriologist in the Dental School of the University of Pittsburgh.

[2] Bödecker, C. F. The bacterial invasion of the enamel in dental caries. *Dental Cosmos*, 1927, lxix, pp. 987, 1002.

FIG. 1. CARIOUS ENAMEL FROM A SHALLOW CAVITY

BP—Bacterial plaque. *E*—enamel. *BC*—bacterial channels. Note the great irregularity of the enamel at the margins of the plaque. The enamel near the center is honeycombed by channels filled with bacteria (BC). Enlargement: approximately 800 ×

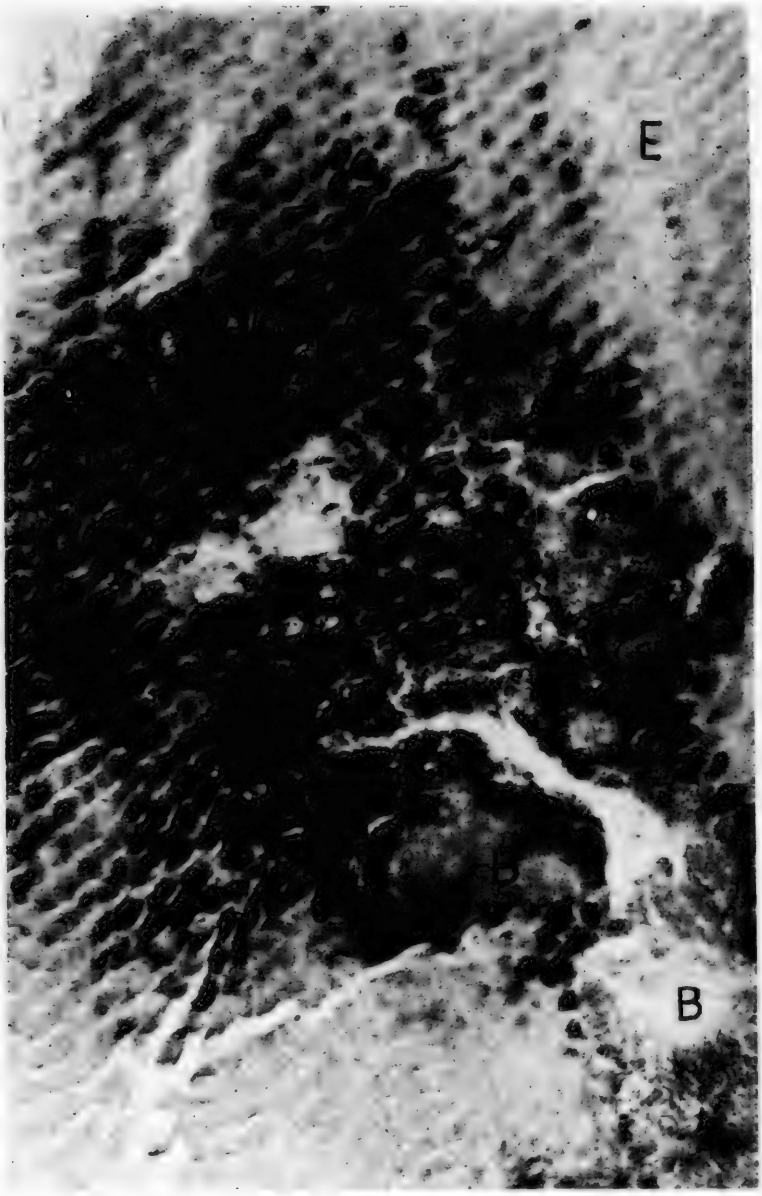

FIG. 2. PENETRATION OF DECAY SURROUNDED BY SOUND ENAMEL

E—Enamel. *ZCD*—zone of carious degeneration. *B*—confluent bacterial foci. Enlargement: approximately 1000 ×

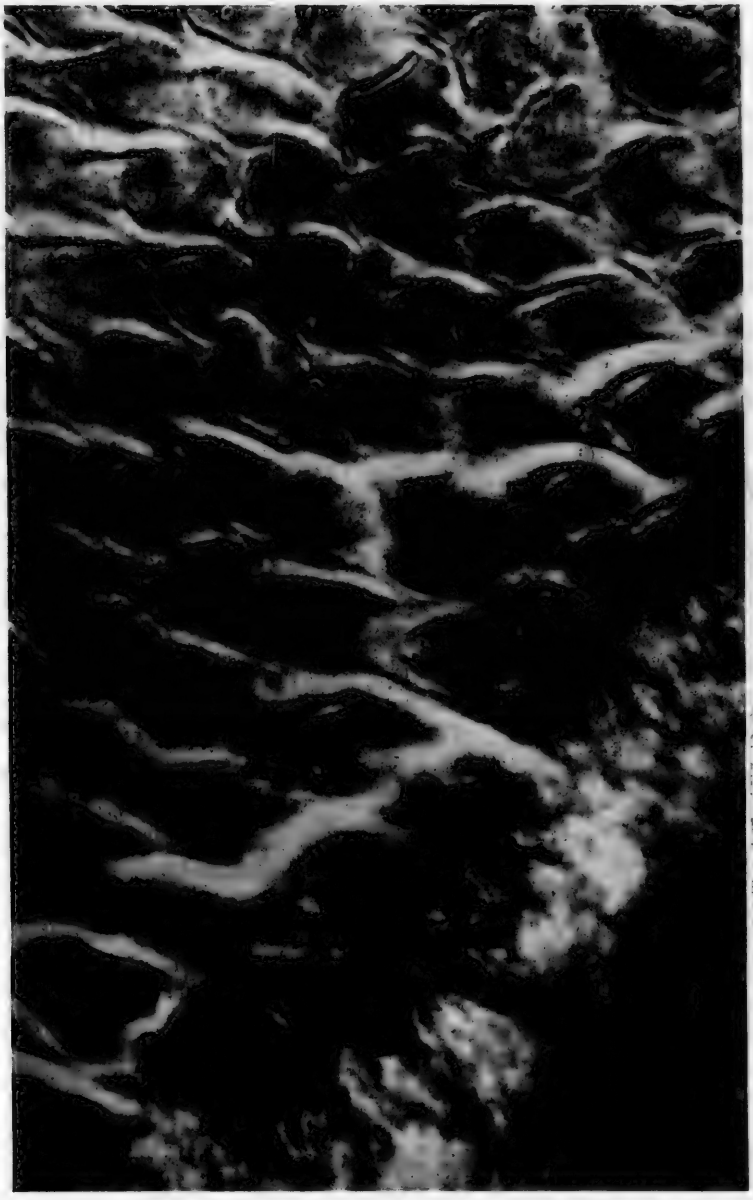

FIG. 3. Enamel (*E*) at the margin of a carious cavity, showing the zone of carious degeneration (*ZCD*) and a bacterial plaque (*BP*) made up apparently of filiform types. Enlargement: approximately 3500 ×

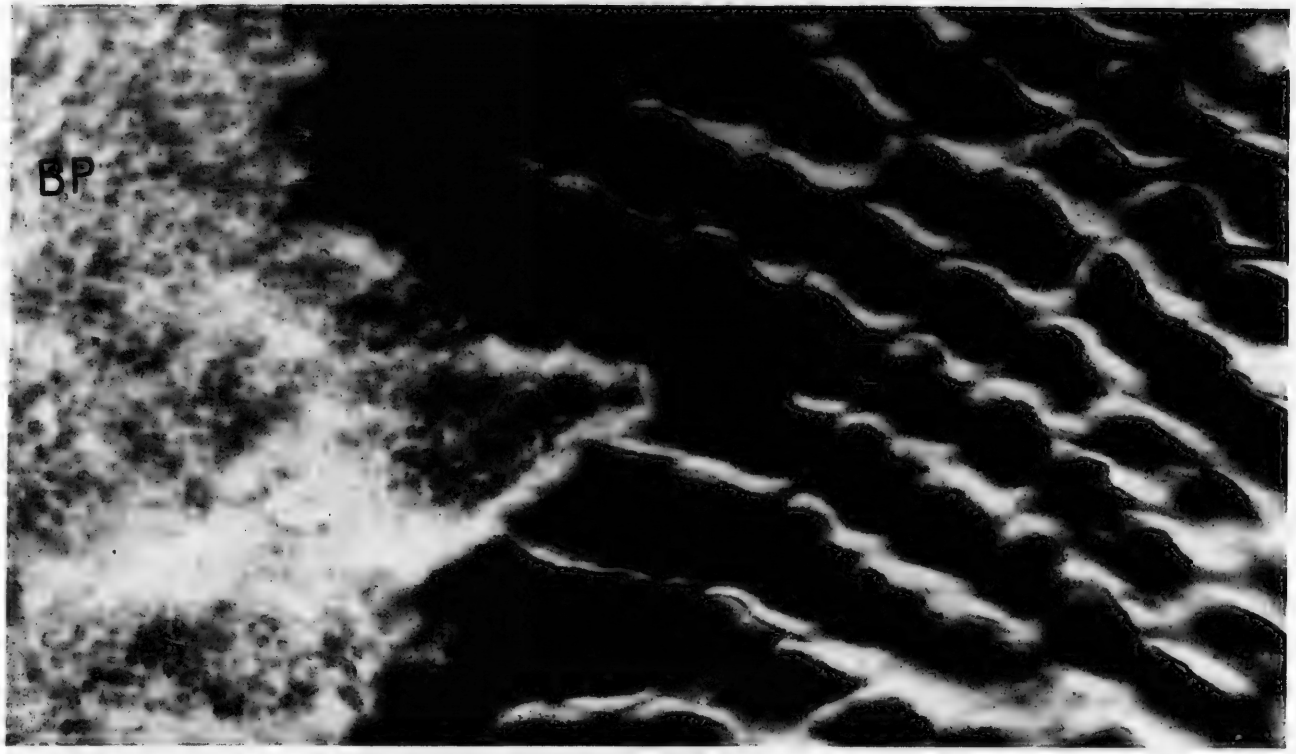

FIG. 4. Carious enamel (*E*), showing only the zone of carious degeneration and a part of a bacterial plaque (*BP*). This appears to be composed solely of cocci. Enlargement: approximately 3500 ×

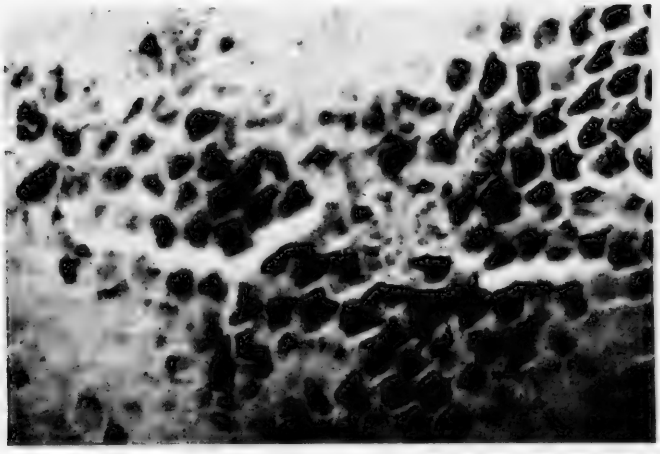

FIG. 5. Enamel showing the normal structure of rods in transverse section (light, indistinct, and honeycomb-like), and the zone of carious degeneration (black cores bounded by light enamel-rod sheaths). A number of confluent bacterial foci can be seen in different parts of the field. Enlargement: approximately 1000 ×. See also *fig. 6*

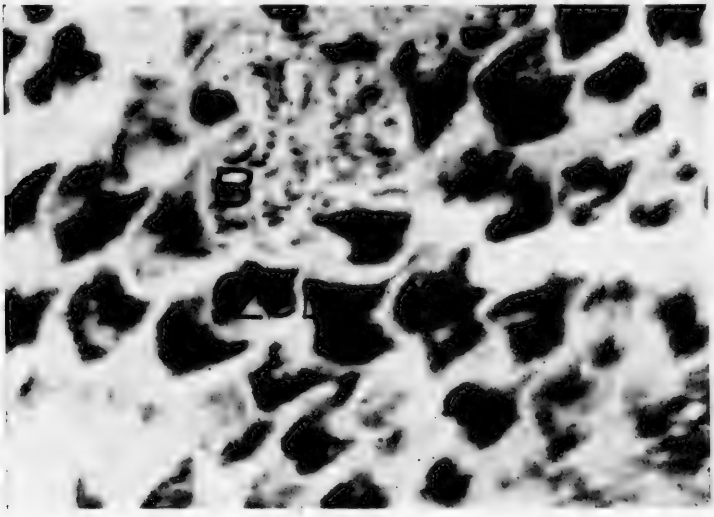

FIG. 6. Same as *fig. 5* under an approximate enlargement of 2,300 ×

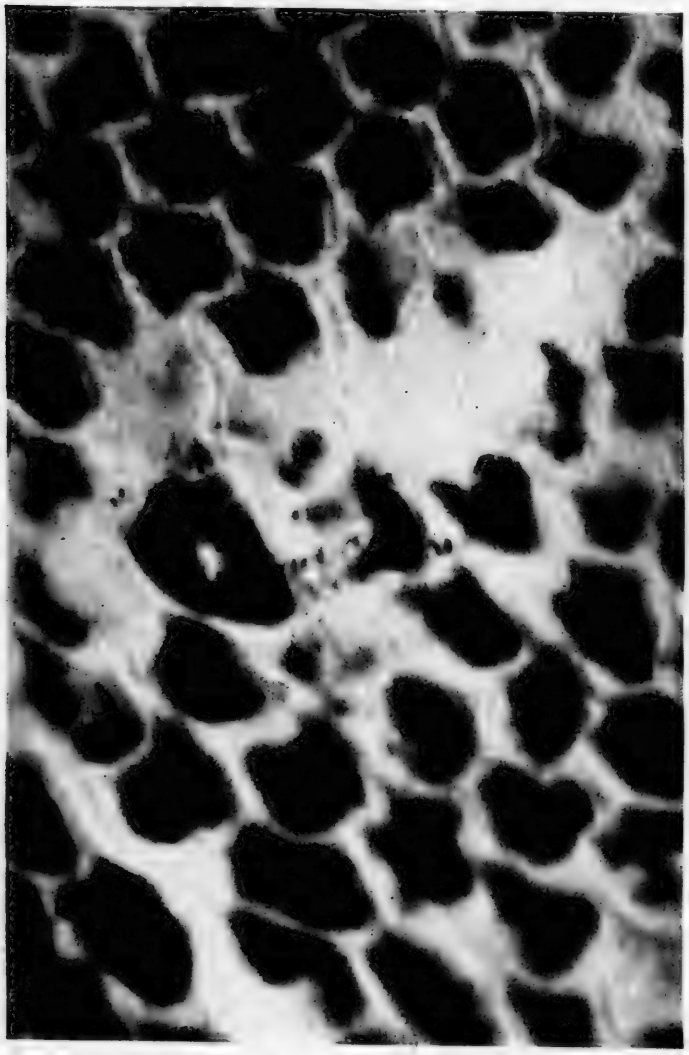

FIG. 7. Individual bacteria (diplococci?) in a zone of carious enamel. Enlargement: approximately 3,500 ×

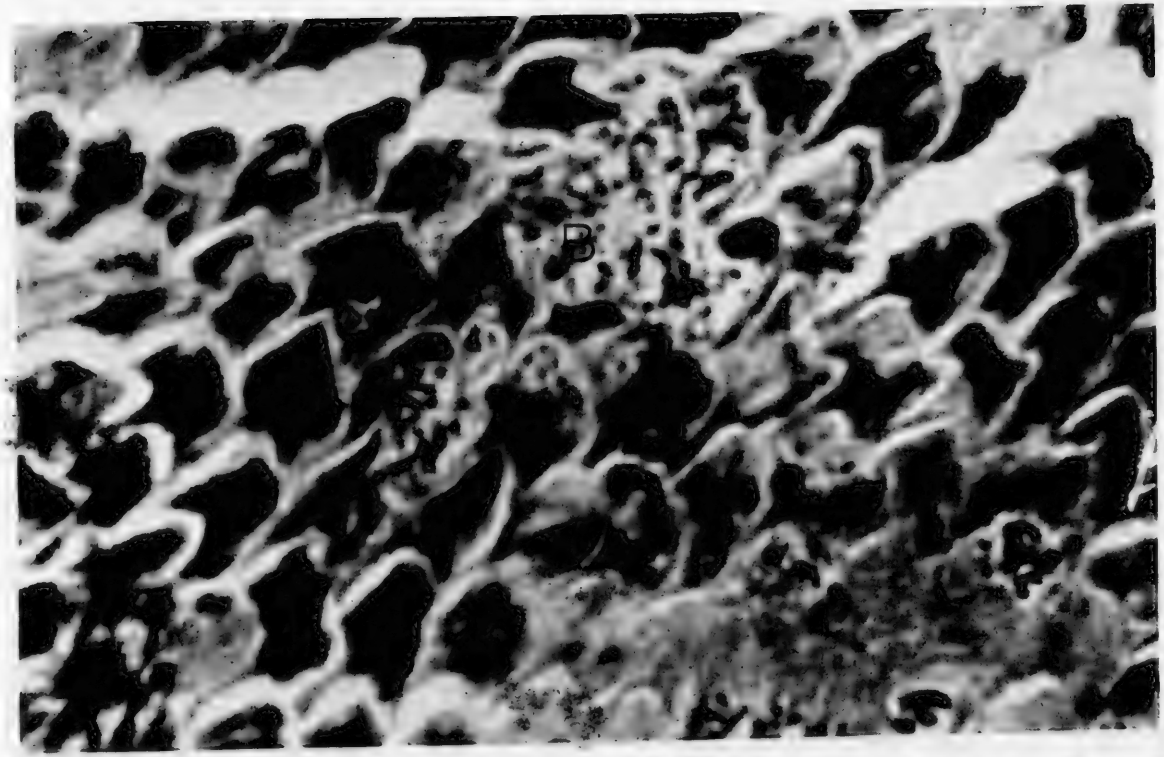

FIG. 8. A small confluent area of bacteria (*B*), with a number of enamel rods in the first stage of infection. These show individual cocci in the body of the rod, leaving the sheath intact. Enlargement: approximately 3000 ×

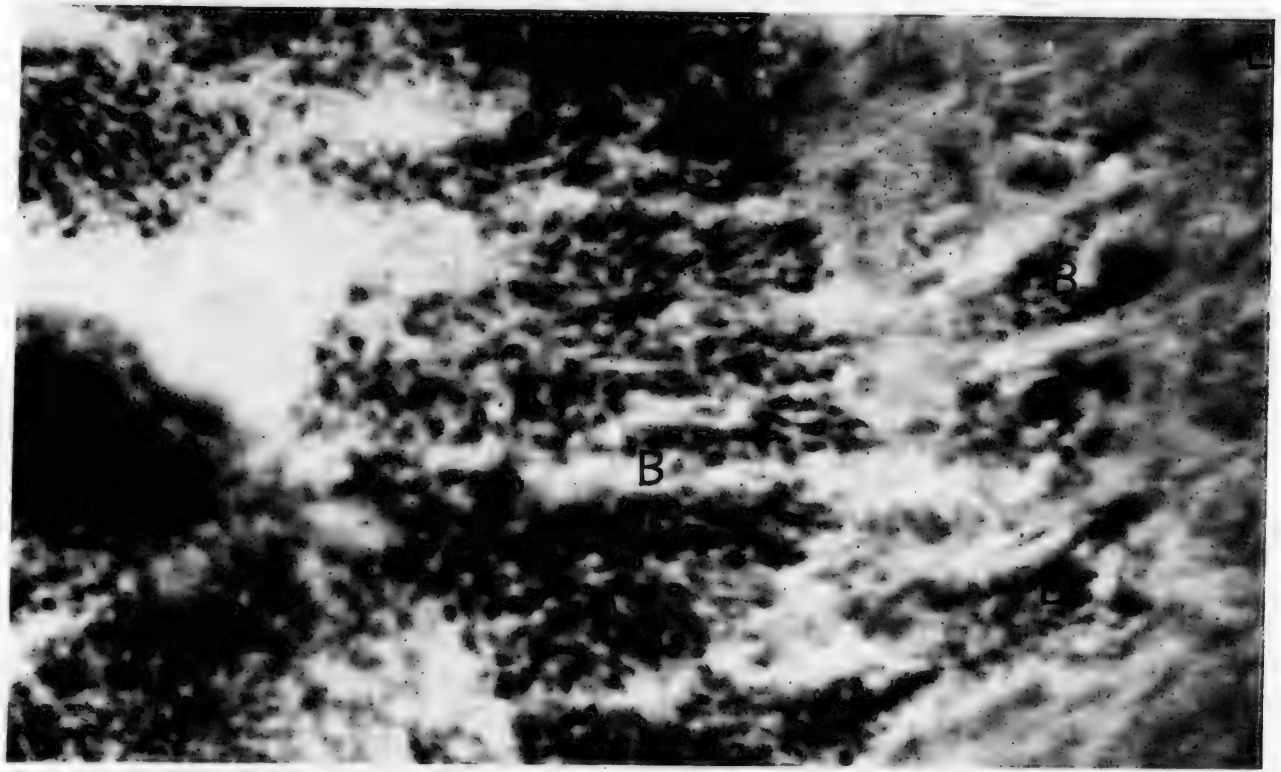

FIG. 9. An oblique section of carious enamel (*E*), showing the penetration of the bacteria (*B*) into the depths. Enlargement: approximately 4000 ×

FIG. 10. Enamel rods in oblique transverse section completely filled with bacteria at *B*, leaving in most places the enamel-rod sheaths intact. Enlargement: approximately 4,000 ×

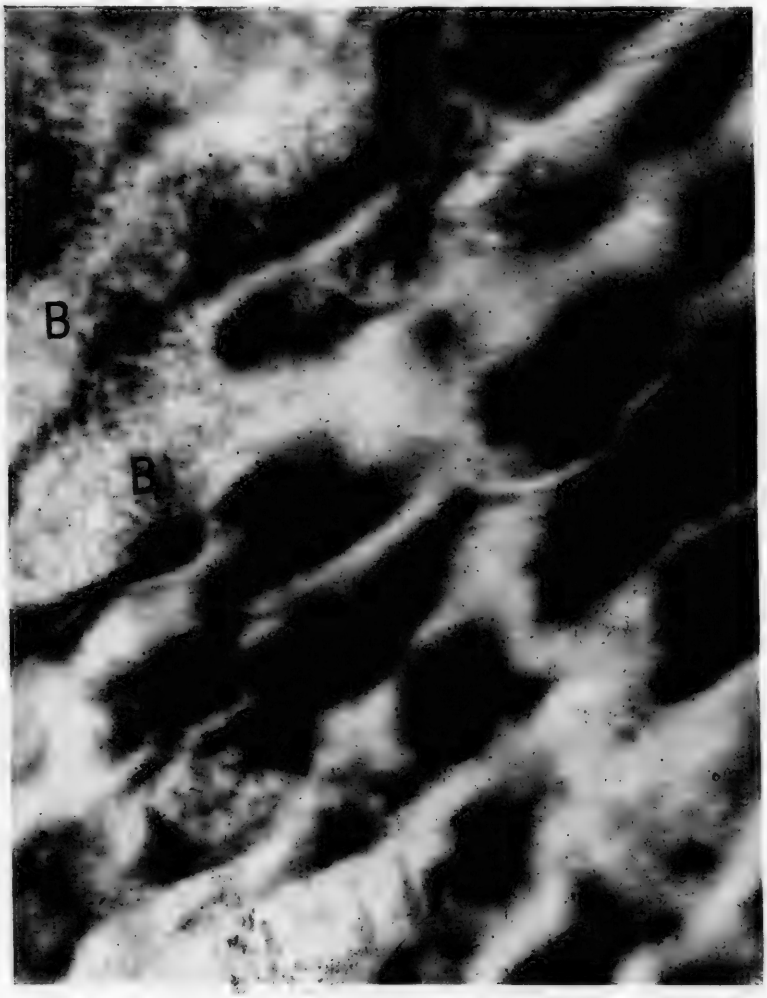

FIG. 11. Confluent bacterial areas, showing the last stage in the destruction of a portion of the enamel-rod sheath between the two *B* rods. Enlargement: approximately 5,200 ×

FIG. 12. An exceedingly high enlargement (8,000 ×) of infected enamel rods (*B-B*), proving that the contents of the rod are destroyed *before* the enamel-rod sheath (*S*) is affected. The latter can be seen as a boundary line between the individual rods

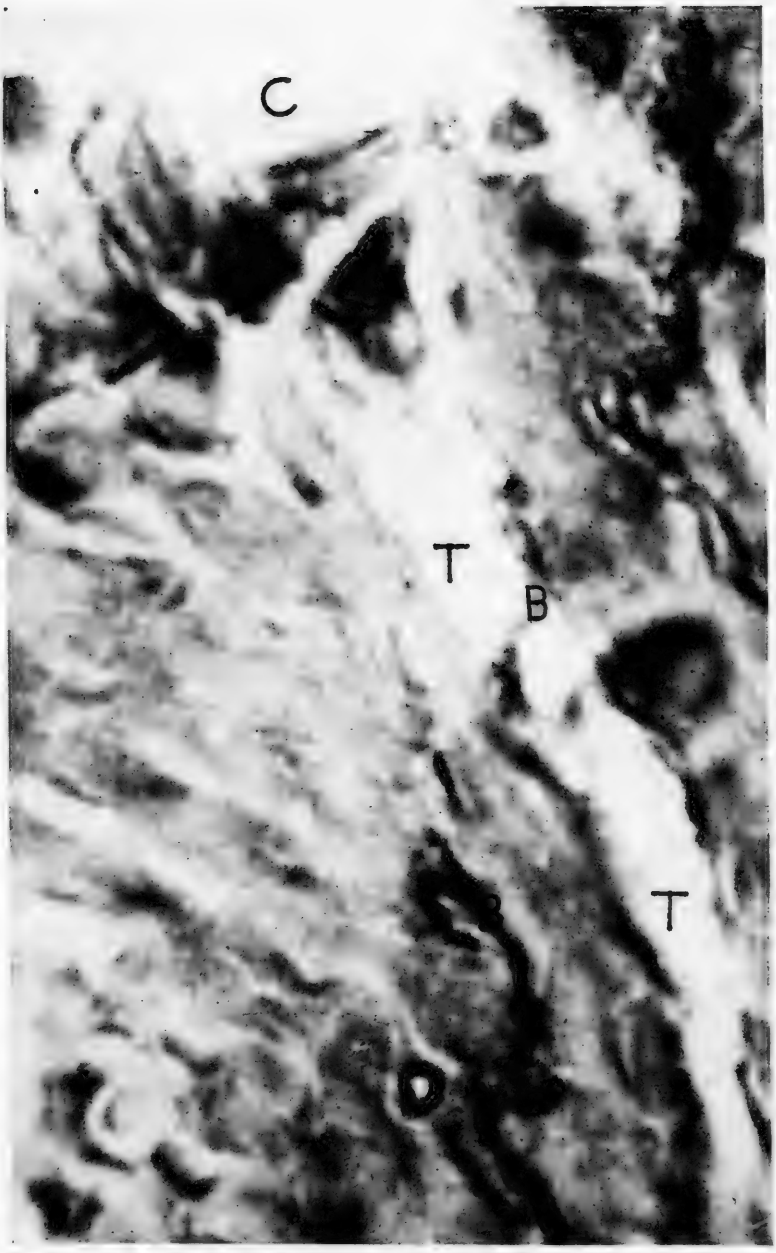

FIG. 13. Fairly normal enamel, obliquely sectioned, showing only at the left upper corner an area of carious degeneration. The somewhat granular line (*B-B-B*) shows bacteria penetrating into a crack in the enamel, probably while the tooth was still in function. In this exceptional case the germs penetrated the enamel *between* the rods, i.e., along the cement substance. At the middle *B*, a tear in the specimen has stretched the bacterial thread across this crevice. *C*—cavity. *TT*—tear in the decalcified organic matrix. Enlargement: approximately 4,000 ×

that the bacteria are in close contact with the organic matrix of the enamel. The suggestion that they are only in chance apposition to the enamel, i.e., that they have been forced into spaces previously formed by the acid agents of decay, seems untenable. A careful examination of these specimens of carious enamel shows that the bacteria are in close contact with the organic matrix, not only in areas which are in a direct line of communication with the surface of the tooth, but also in the most remote undercuts of the cavity. *Fig. 1* shows a condition of elaborate honeycombing of the enamel, the microscopic channels ranging in size from the width of a score down to the width of a single enamel rod (4μ). Therefore it does not seem possible that pressure from mastication can be alone responsible for the presence of bacteria in these remote depths of the cavity, but the supposition that the bacteria proliferate in the presence of nourishment and thus penetrate the enamel so deeply is reasonable. We believe that these bacteria subsist upon the organic matrix of the enamel, but there appears to be a difference in the resistance of the various structures of this tissue to their attack. We have consequently come to the conclusion that there are two kinds of protein in the enamel; namely, a keratin and an albumin.

This deduction is warranted by the local differences in the penetration of the bacteria into the enamel. The specimens clearly show that the bacteria proliferate as well as penetrate deeper into the core of the enamel rod, thus leaving the enamel-rod sheath intact for some time and indicating that the latter is more resistant to bacterial destruction than the content of the rod upon which the bacteria apparently subsist. This difference in the resistance of these two structures indicates that they are chemically different, and that in all probability the enamel-rod sheath is a keratinous material while the enamel rods contain albumin-like material.

Professor Gies'[3] preliminary inquiry into the chemical nature of the organic matrix of the enamel showed that it contained protein. He is endeavoring, with the aid of Dr. Theodor Rosebury, Second Gies

[3] Bödecker, C. F., and Gies, William J. Histochemical proof of the presence of protein matter in dental enamel. *Proceedings of the Society for Experimental Biology and Medicine*, 1924, xxii, p. 175. Also Gies, William J. Direct evidence of the presence of albuminous matter in enamel. *Journal of Dental Research*, 1926, vi, p. 143.

Fellow in Biological Chemistry at Columbia University, to determine the nature of the protein or proteins in this matrix.

The observation that the structures of the enamel offer a varying resistance to attack by bacteria strengthens the theory that the enamel is formed by the direct transformation and calcification of the ameloblasts. If this is true, then the walls of the ameloblasts are transformed into the enamel-rod sheaths, while the content of the cells is calcified to form the body of the enamel rod. This would therefore result in the production of two different protein masses in mature enamel, that is, the *keratinous* enamel-rod sheath and the calcified *albumin* or *albumin-like* content of the rods. When therefore the mineral matter is removed from enamel by the acid of decay, the organic matter remains, exposing the albumin in the rod to the direct destruction that bacteria promptly bring about.

Summary

(1) Enamel is destroyed during dental caries by two agencies: (a) by acid, in all probability lactic acid, formed by B. acidophilus, and (b) by the proteolytic action of bacteria (streptococci or diplococci?) upon a portion of the protein content.

(2) The selectivity shown by the proteolytic bacteria, in destroying the content of the enamel rod more quickly than the enclosing enamel-rod sheath, suggests that these two structures differ chemically. The authors believe the sheath is composed of keratin and the rod proper of calcified protoplasm (albumin; albumin-like material).

(3) If this theory is correct, it suggests that the enamel is developed embryonically by the direct calcification of the enamel-forming cells, the ameloblasts, and that the enamel is therefore a calcified tissue.

Technique of preparing specimens

Carious enamel was decalcified by the acid-celloidin decalcifying method,[4] and sectioned after treatment with the double embedding process (paraffin-celloidin). An important factor for the detection

[4] Bödecker, C. F. Fundamentals of Dental Histology and Embryology, 1926, p. 224. (The proper solvent of the celloidin for this decalcifying method is methyl alcohol, *acetone free.* By a misprint in this book it is stated that "methyl alcohol, acetone" should be used.)

of bacteria in the tissue is the production of exceedingly thin sections. To date the double embedding process has been the only one by which it was possible to obtain 3μ sections of the organic matrix of the enamel. These thin sections were stained with Heidenhain's ferric hematoxylin, which develops a bluish-black stain with delicate gradations. Considerable experimentation is necessary to achieve just the correct differentiation in order to show the bacteria.

General note regarding the illustrations[5]

Normal enamel is markedly differentiated from carious enamel, the former taking the stain with greater difficulty. A novel and interesting factor in these specimens is the occurrence of a zone between normal and diseased enamel that takes the Heidenhain ferric hematoxylin stain most intensely. This area, which is noted as an intensely dark zone, separates the normal from the infected tissue and appears to be a form of degeneration of the organic matrix preparatory to its destruction by the bacteria. This zone has never been seen before (as adult enamel can not be decalcified by ordinary methods), and the authors have named it the "*zone of carious degeneration*," designated in the illustrations as "*ZCD*." The illustrations are reproductions of photomicrographs made with a Zeiss apochromatic immersion objective of 1.5 mm. focal length and various compensating eyepieces.

[5] The photomicrographs in *figs. 5* to *8*, inclusive, were made by Else Barkusky of the Lette House, Berlin, Germany.

A STUDY OF SOME OF THE MATERIALS USED IN THE CASTING PROCESS[1]

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CONTENTS

I. Introduction 55
II. Gold 56
III. Wax 64
IV. Investment 67
V. Semi-indirect method 69

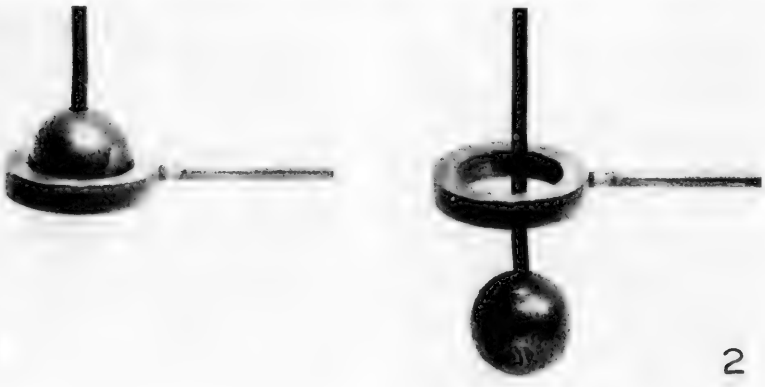
I. INTRODUCTION

I feel that I owe an apology for selecting as the subject of my paper some questions concerning the casting process, knowing the long list of able students and conscientious workers in this field who have already addressed this Society on the same subject. But as most of us are daily confronted with certain problems of casting, renewed study of some of the difficulties we frequently encounter may prove helpful, I hope, in our every-day work. The first years after the introduction of the casting process were almost entirely taken up in an effort to master its technique, and any unsatisfactory results were laid to faulty manipulation. But even today, after the close of this preliminary experimentation, many conscientious workers have found that changes take place during the process of casting which cannot be explained through lack of skill of the operator nor through the fault of the casting device, but which are due to the physical properties of the materials employed. Let us consider how some of these changes can impair the exactness of our work in spite of the most painstaking care on our part.

[1] Read at a meeting of the First District Dental Society of the State of New York, New York Academy of Medicine, January 4, 1926.

II. GOLD

We all know the physical law governing the expansion and contraction of metals through changes of temperature. From our school days we remember the instrument that demonstrated this fact in a most striking manner: a metal ball too big to pass through a certain metal ring easily passed when the ring was heated; as illustrated in *figs. 1* and *2*. We also learn in the text books that different metals have different coefficients of expansion. We are surprised to find that the coefficient of expansion of gold, the metal most used in our work,



Figs. 1 and *2*. Demonstrate the expansion of metal. The metal ball is too big for the ring in *fig. 1*, but passes easily when the ring is heated (*fig. 2*).

has not yet been definitely established. Landolt and Bornstein give it as being 0.000014. A. Miller gives 0.000015. W. A. Price puts it at 0.000022. The disagreement in these figures may be due to differences in the ranges of temperature at which the experiments were made, for the coefficient of expansion increases as high temperatures are attained. Since we always have to deal with temperatures reaching the melting point of gold, L. J. Weinstein gives a coefficient of 0.000017, that is to say, for every degree centigrade there is a linear expansion of 17 millionths of the length; i.e., for one meter which is 1000 mm. it expands 17 millionths of its length per 1°C., which is 0.017 mm. and for 1000°C.—about the melting point of gold—it

expands a thousand times as much, which is 17 mm. or 1.7 per cent. For one inch it would be 17/1000 of an inch. If a tooth is 1/3 of an inch in mesio-distal diameter, 5/1000 of an inch is the amount of expansion we have to deal with. These changes take place while the metal is in its solid form. When gold is melted and changes its state, it expands another 4.13 per cent by volume, which is a linear expansion of 1.64 per cent (*fig. 3*).

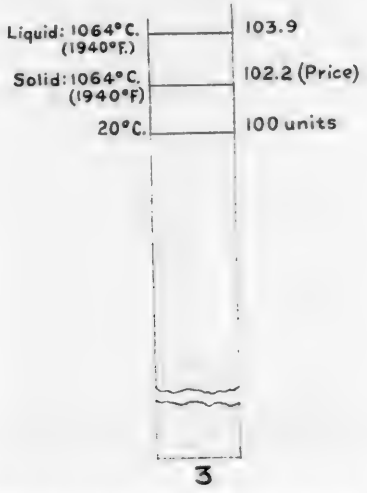
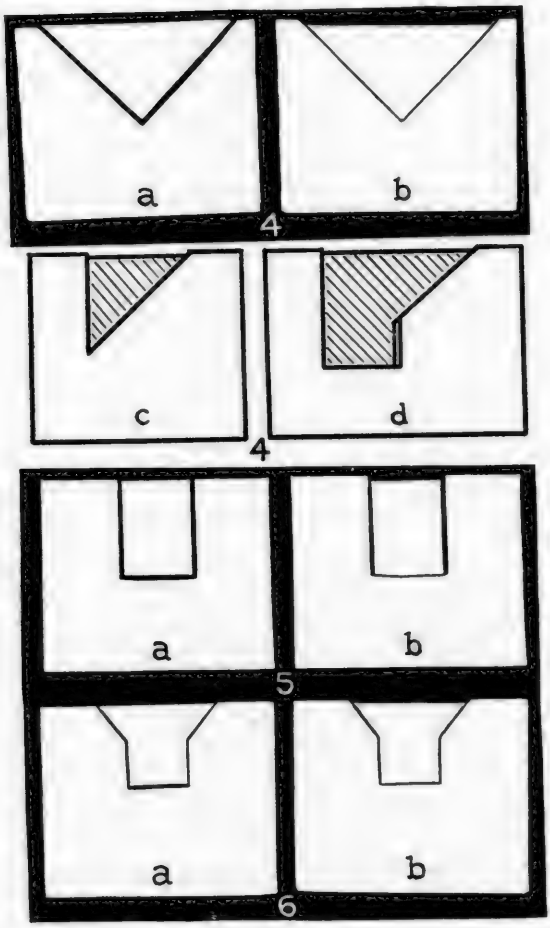


Fig. 3. Represents a bar of gold of 100 units. The line at 20°C. shows its length at room temperature; the middle line, the amount of expansion at the melting point; the upper line, the amount of expansion after the gold has been liquefied.

This great change however, which occurs in passing from the liquid to the solid state, is overcome in the process of casting. As the gold solidifies, we add new gold to fill the mould, and it is only when the gold remains liquid within the mould and further addition ceases that this contraction is manifested. We then have the so-called "pipes" near the sprue. This generally occurs in large castings where the bulk of the metal retains enough latent heat to stay liquid. It is evident that by making thin castings, using a cold mould, and not overheating the gold, this difficulty can be overcome. It is only the difference



Figs. 4, a, b, c, d. Diagrams of conc-shaped cavities, where the contraction of the gold casting would not prevent a perfect result. See *figs. 5, a, b.*

Figs. 5, a, b. The same as *figs. 4, a, b, c, d,* for cylindrical cavities. See *figs. 6, a, b.*

Figs. 6, a, b. The same as *figs. 4* and *5,* for a combination of conical and cylindrical types of cavity.

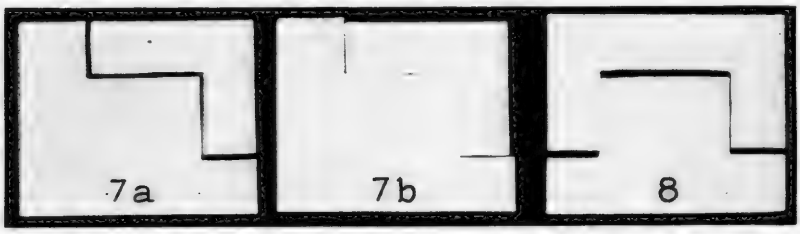
between the temperature of melting and of the room that causes the contraction so detrimental to some of our work. In the construction of a steel-girder bridge, for example, allowance has to be made for the expansion of the steel. In the great bridge over the Firth of Forth, in Scotland, which is over a mile and a half long, the total expansion amounts to six feet. There the difference between summer and winter temperatures is not over 50°C., while in our work we deal with a temperature that exceeds 1000°C. The melting point of gold being 1064°C., which is often increased in some of our alloys through the addition of platinum, I can safely make the statement that for this very reason every cast we make is smaller than its pattern, for the gold enters the mould at a temperature of 1064°C. (1940°F.) and then is allowed to cool to room temperature, causing (even for our comparatively small work) quite an appreciable amount of contraction.

It is not within the scope of this paper to discuss cavity preparation as such, although I fully realize its great importance for the casting technique. Let us examine only the factors which interfere with perfect adaptation of the inlay. Of course, undercuts in the cavity are detrimental to a perfect result. If the wax is hard, it breaks in the attempt to remove it from the cavity. Softer wax may spring open, enough to disengage from the cavity, or it may even bend out of shape. In either case of course a perfect adaptation is impossible. Another important detail in cavity preparation is the need for absolutely smooth walls, as imperfections in their surfaces change their respective places and prevent perfect seating of the inlay.

In order to see where and when the contraction of gold interferes with a good result of the casting, let us divide the work into four different groups. Let *Group I* comprise inside cavities, illustrated by *figs. 4, 5,* and *6*. If it were possible to make a cone-shaped cavity as illustrated in *figs. 4, a, b, c, d,* no amount of contraction would prevent a perfect adaptation of the casting to the cavity walls. There would be a shyness only at the cavity margins. In a cylindrical cavity, as presented in *figs. 5, a, b,* contraction would result in a slight space at the cavity walls and a shyness at the cavity margins. *Figs. 6, a, b,* illustrates a combination of conical and cylindrical types of cavity with the same results pertaining to each, i.e., shyness at the margins, but perfect adaptation to the walls in the cone-shaped cavity. The

contraction of the gold causes *no* difficulties in cavities belonging to Group I because these cavities are always very small and therefore discrepancy due to contraction is very slight. The casting always goes to place, and a small amount of polishing at the margins makes a perfect inlay. I want to emphasize however the importance of a broad bevel in securing perfect adaptation.

In *Group II* we shall consider an M. O. cavity as the representative type (*figs. 7, a, b*). Here we find perfect adaptation at the pulpal and axial walls, but a slight space at the gingival and disto-occlusal margins owing to contraction. The buccal and lingual mesial margins, and the buccal and lingual occlusal margins, show perfect adaptation.



Figs. 7, a, b. Illustrate the contraction of a cast M.O. inlay, and the resultant slight space at the gingival seat.

Fig. 8. Diagram of an M.O.D. inlay, or full crown, where the contraction of the gold proves disastrous.

A slight apron at the gingival, and the polishing of the disto-occlusal edge, may help to overcome these difficulties and make a useful inlay. But a better way is the technique advocated by Drs. Mortonson and Mortonson. A slight addition of softer wax is made at the gingival part of the wax pattern not only to assure a better impression of the gingival seat, but also to secure a perfect adaptation at that point. We see that in castings belonging to Group II, with the right mechanical help, any difficulties caused by the contraction of the gold can easily be overcome and perfect results obtained.

In *Group III* we shall consider a type of casting typified by an M. O. D. inlay (*fig. 8*). In this class of work any contraction in the casting causes serious difficulties unless measures to compensate for the shrinkage of the gold are adopted. We can readily see that, if the

two axial walls are perfectly parallel and the distance between the corresponding axial surfaces of the casting is shorter than in the tooth, the inlay will not go to place. And even if the axial walls of the cavity converge decidedly toward the occlusal, there will still be a marked space at the gingival seat. Through trigonometry we could easily

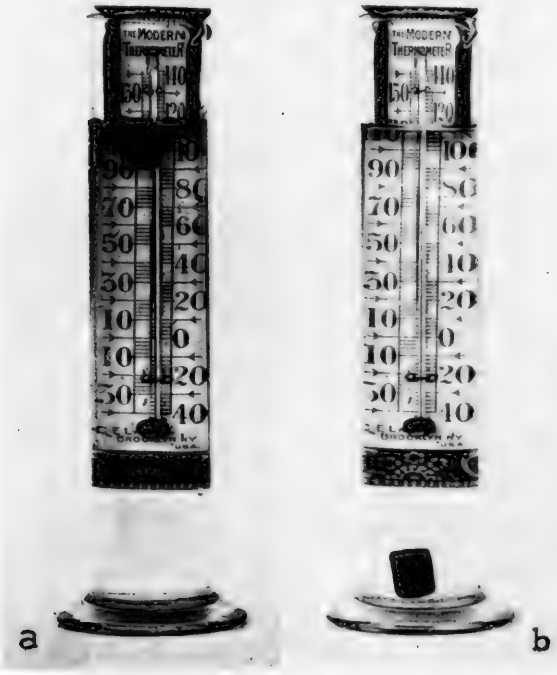


Fig. 9. Demonstrates the expansion of wax by heat. In *a*, the dark piece of wax is at the bottom of the water at 60°F. When heated to 88°F., the wax expands enough to float, as shown in *b*. In this experiment, the wax must be loaded.

prove that the space increases in proportion as the axial wall approaches a perpendicular to the base of the tooth; in other words, as the two axial walls approach the parallel. Here again the technique introduced by Drs. Mortonson and Mortonson is of great help, as the additional wax at the gingival border not only secures a perfect

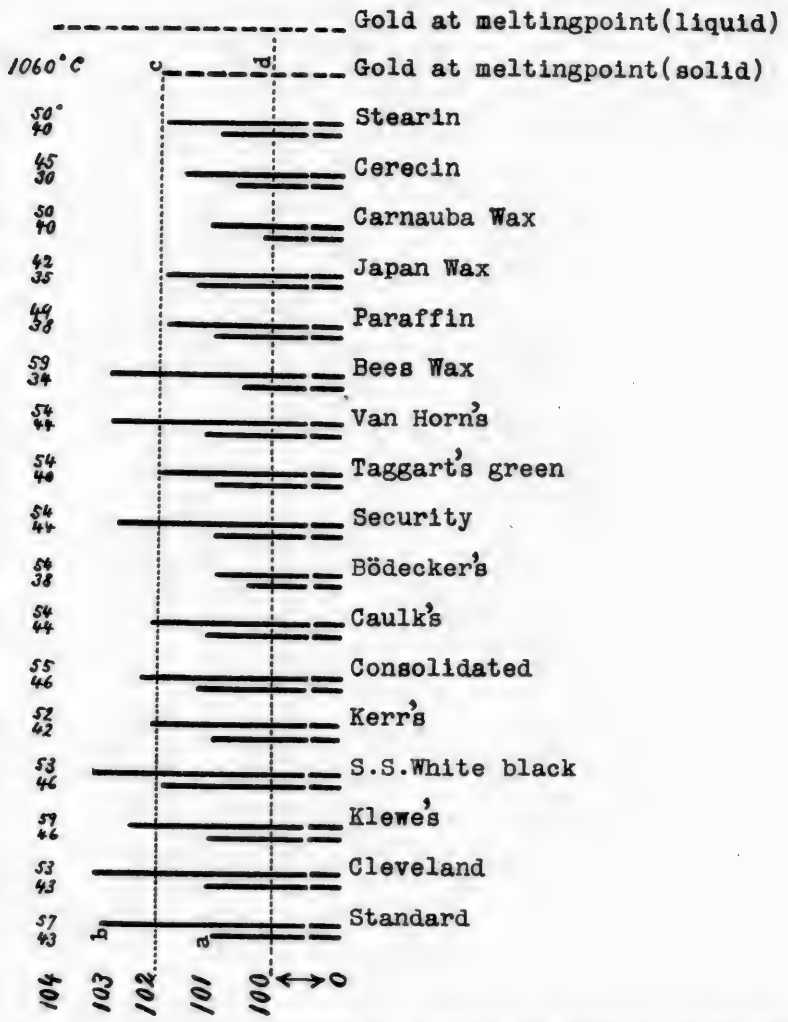


Fig. 10. Data relating to different waxes, showing their expansion at the respective working temperatures, compared with the expansion of gold.

a—lowest working temperature. *b*—highest working temperature. *c*—expansion of gold at the melting point. *d*— ⟷ 100 units at room temperature.

impression at that point, but also slightly increases the length of the approximal flanges and, under favorable conditions, even widens the space between the axial walls, thus giving very good results in many cases. Thin castings or inlays of soft gold may, through pressure or tapping, be bent sufficiently and forced into place, but this is almost impossible with heavy castings or where a hard-gold alloy is used. An apron at the gingival border would not help much in this type of

TABLE 1

Data showing the degree of expansion of compressed and cooled waxes, after heating

WAX	COMPRESSED AND MEASURED AT 20°C.	HEATED TO MAXIMUM AND MEASURED AT 20°C.	EXPANSION
no.	*mm.*	*mm.*	*per cent*
1	2.80	3.75	33.93
2	2.70	3.65	35.19
3	2.10	2.65	26.19
4	2.58	3.30	27.91
5	1.95	2.25	15.38

TABLE 2

Data showing the degree of contraction of stretched and cooled waxes, after heating. (See fig. 11)

WAX	DRAWN AND MEASURED AT 20°C.	HEATED TO MAXIMUM AND MEASURED AT 20°C.	CONTRACTION
no.	*mm.*	*mm.*	*per cent*
1	50.00	48.30	3.40
2	50.00	40.40	19.20
3	50.80	44.65	12.11
4	50.60	39.10	21.34

cavity, as the approximal flanges are nearly always bent away from the axial walls in an effort to seat the inlay. We must not be deceived by perfect occlusal edges in these cases nor take for granted that the gingival borders, which may be hidden under the gum, are equally good. It is in this Group III that for the first time we encounter difficulties caused by the contraction of the gold—difficulties easily overcome by a method that enlarges the whole wax pattern, as we shall see later.

In *Group IV* the full crown is the representative type. All the

difficulties encountered in Group III are here greatly exaggerated, as no stretching or bending of the metal is possible. The same fact remains true: the more the axial walls approach the parallel, the greater the difficulty in seating the crown. But even in a very conical preparation, a perfect adaptation is impossible unless the wax pattern is enlarged before casting, the details of which we shall consider later.

III. WAX

Of course wax, like other materials, is expanded by heat. This can easily be illustrated by the experiment shown in *fig. 9*. A piece of wax lying at the bottom of a receptacle filled with water at a certain temperature rises and floats on the water when the temperature is raised, showing that the volume of the wax has increased. It is evident that in this experiment the wax may be heated only to a degree that does not endanger the maintenance of its shape.

Fig. 10 lists a number of waxes investigated by W. A. Price, and indicates their respective working temperatures and the percentages of their expansion. If, for instance, we take Kerr's inlay-wax and heat it to 42°C., there is about 1 per cent expansion partially to compensate for the contraction of the gold. If S. S. White black wax is heated to 46°C., there will be about 2 per cent expansion according to Dr. Price's findings. It is evident that we have to stay below the lowest working temperature, as otherwise the wax would get soft and lose its shape. In order to take advantage of the expansion of the wax, it would be necessary to invest the wax pattern in an investment of the desired degree of heat and keep it at that temperature until it sets. This, however, is not quite as easy as it may seem, for we have to deal with a very annoying factor; namely, the internal strain or the so-called latent elasticity imparted to the wax by the manipulation. This strain or elasticity manifests itself when the wax is heated to or near its working temperature. When, for instance, the wax is compressed and cooled, it expands to an enormous degree if heated, as shown in *Table 1*. On the other hand, if the wax is stretched and cooled, it contracts again when heated (*fig. 11* and *Table 2*). When wax is bent and then heated, it tends to a very marked degree to return to the original shape, as shown in *figs. 12, 13,* and *14*.

Therefore, when we want to avail ourselves of the fact that heat

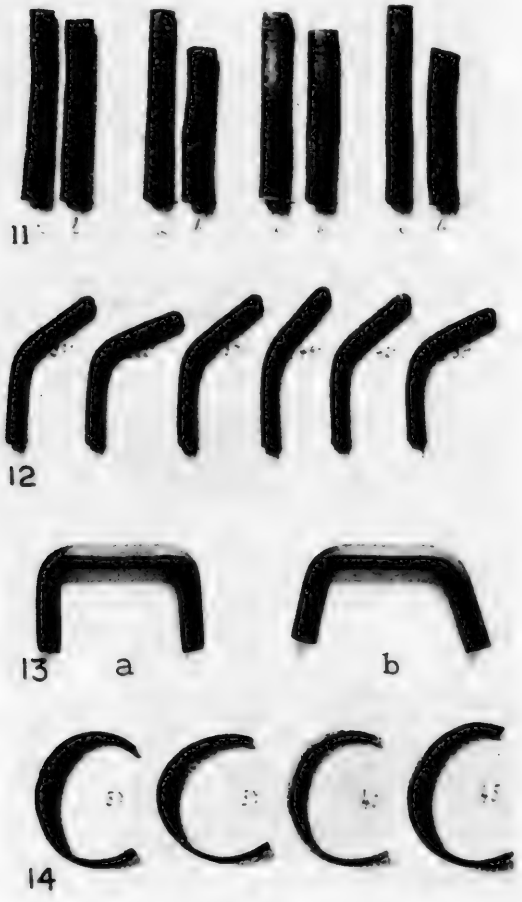


Fig. 11. Contraction of four different waxes drawn out and cooled. *a*, shows the original size; *b*, was heated to the working temperature.

Fig. 12. Bars of six different waxes had been bent at right angles. They all bent back somewhat after being heated.

Fig. 13. Wax bar. *a*—before heating; *b*—distorted by heat.

Fig. 14. Closed rings of four different waxes opened by heat.

expands the wax, this latent elasticity must be eliminated, otherwise our wax pattern would be ruined. This can be done by heating the wax to its working temperature while it is in position, thus giving the molecules freedom to arrange themselves in their natural unrestained relation to one another. If we press the wax into a cavity and even ask the patient to bite on it, there is imparted to it a great amount of

Fig. 15. Wax pattern. *a*—before heating; *b*—distorted by heat

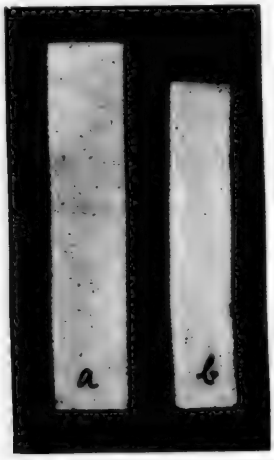


Fig. 16. Plaster. *a*—before heating; *b*—contracted by heat from a blow-pipe

latent elasticity or internal strain, which is maintained as long as the wax is kept cold, but if it is warmed after being removed from the cavity, it changes its shape (*fig. 15*). We must remove all internal strain from the wax while it is in the cavity by warming it with hot water, warm air, or a hot instrument, and readapting and rewarming it until it retains its shape. Thus prepared, we can cool it for removal

and it will now evenly expand if flasked in warm investment. This method, however, has its drawbacks and its limitations. We have to apply carefully the right amount of heat, work rapidly as the heat hastens the setting of the investment, and be careful not to alter the shape of the wax in applying the warm investment. In other words, we must master all details of the technique. Even then the method is limited to cases where an equal expansion at all points of the wax pattern is needed, which very rarely is the case.

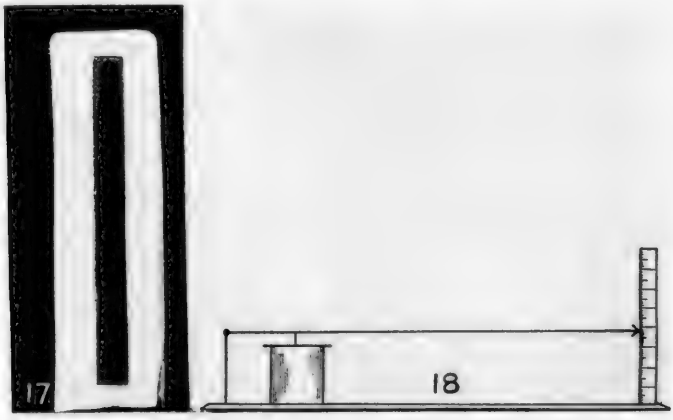


Fig. 17. Brass bar embedded in plaster. Space between end of metal bar and plaster indicates amount of expansion of plaster.

Fig. 18. Instrument for determination of the time at which expansion of plaster takes place.

IV. INVESTMENT

Changes in the investment may also be the cause of considerable diminution in the size of the finished cast. The main constituents of the investment are silex and plaster. Silex is not deteriorated by heat but plaster when heated shows very marked contraction, which varies with the degree and duration of the heat. *Fig. 16, b* shows the diminution in size of a piece of plaster, after being heated with a blow-pipe, compared with its original size (*fig. 16, a*).

Another important physical property of plaster is its expansion during the process of crystallization, which can easily be measured

with a micrometer. *Fig. 17* shows a brass bar embedded in plaster. The space between the plaster and the end of the brass bar indicates the expansion of the plaster. *Fig. 18* shows an instrument with which this expansion can be not only measured, but also its beginning and duration observed. We find that the expansion sets in at the beginning of crystallization and ends when the process of hardening is

TABLE 3

Data showing expansion of investments after setting

	PARTS				
Plaster	1	1	2	3	4
Silex	2	1	1	1	1
Expansion (per cent)	0.10	0.20	0.34	0.38	0.40

TABLE 4

Data showing contraction of investments after elimination of wax

SILEX	PLASTER	CONTRACTION
parts	*parts*	*per cent*
3	1	0.40
2	1	0.69
1	1	0.80
1	2	0.90
1	3	1.20
1	4	1.34

completed. In the investment a certain amount of expansion also takes place, in the same proportion, for plaster is one of its constituents (*Table 3*).

The investment also shows when heated a considerable amount of contraction, which is proportional to the amount of contained plaster (*Table 4*). *Fig. 19* shows both the expansion and contraction of investments that are dependent upon the proportion of the plaster and temperature. It is therefore very important not to overheat the investment. As long as the investment contains moisture, the temperature will not rise over 150°F. to 212°F. Therefore, the right time to cast is at the moment when the production of steam ceases, thus having an investment with almost no contraction.

The expansibility and other properties of plaster make it a very

valuable material for a model in the semi-indirect method. It is very important to make the plaster as stiff as possible, for the thinner the mix, the less the resistance and expansion. One part of water to two parts of plaster by weight is a very good proportion. *Fig. 20* shows four plaster bars containing the same amount of plaster, 30 grams, the differences being only in the content of water, of which the

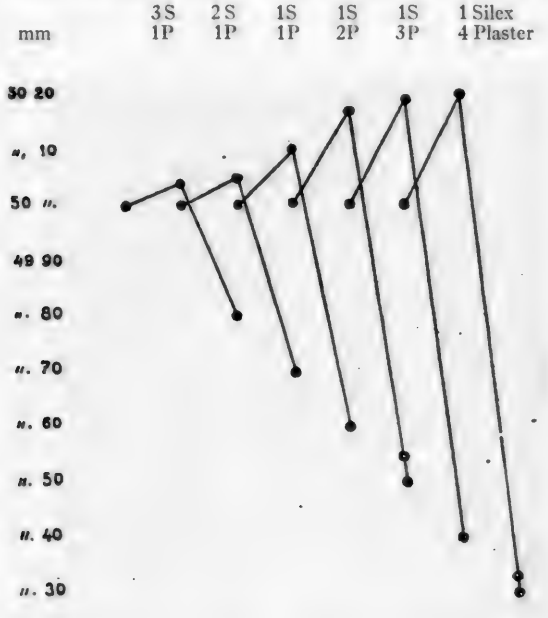


Fig. 19. Showing six different investments, their initial expansion, and their final contraction after heating.

first bar contains 12 cc., the second 24 cc., the third 36 cc., and the fourth 48 cc. It is evident that the shortest bar is the strongest and best fitted for our purpose.

V. SEMI-INDIRECT METHOD

The first step in the semi-indirect method is to select a copper band or, better, an aluminum band of the right size and take an impression with compound, as shown in *fig. 21.* This impression is filled with

plaster and laid aside. In case of a full-cast crown, where a plaster model of maximum expansion is desired, a slow-setting plaster like Kerr's Snow White No. 1 is used and spatulated for two minutes. This prolonged spatulation not only reduces the setting time from 45 minutes to about 5 minutes, but greatly increases the expansion of the plaster. In case of three-quarter crowns, where less expansion is necessary, a quick-setting plaster like Kerr's Snow White No. 2 is indicated. While the plaster is hardening, a rough wax-impression of

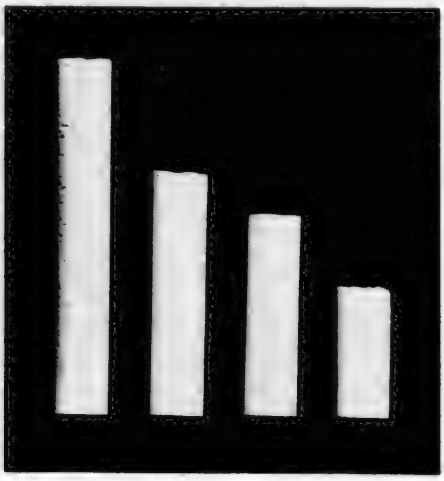


Fig. 20. Four bars of plaster containing the same amount of plaster, but different quantities of water.

the cavity is taken, securing the occlusion and the contact points (*fig. 22*). The plaster model is now freed from its impression material and, while the plaster is wet and warm, the rough wax-pattern is transferred from the mouth onto the plaster tooth (*fig. 23*). No oil or vaseline should be used on the plaster model; if the plaster is wet, wax will not stick to it even if melted on with a hot spatula.

Any carving and correction can easily be done out of the mouth, but the final check up is made on the natural tooth. In this way we have not only the advantage of an indirect method, but also the exactness of the direct method combined with the valuable fact that the

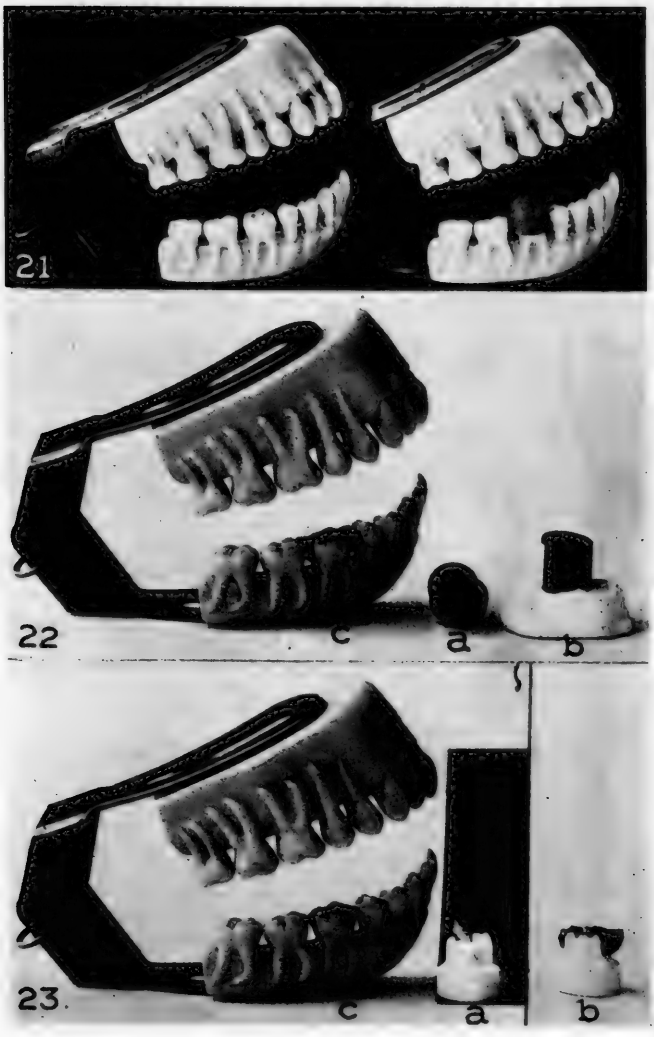


Fig. 21. Taking an impression for an M.O.D. inlay of the lower right first molar. See *fig. 22.*

Fig. 22. See *fig. 21.* *a*—band with compound impression. *b*—impression filled with plaster. *c*—wax pattern securing contact point and occlusion in the mouth. See *fig. 23.*

Fig. 23. See *figs. 21* and *22.* *a*—plaster model. *b*—wax pattern transferred from the mouth to the plaster model. *c*—wax pattern replaced on the tooth in the mouth for final check up after being carved on the plaster model.

wax has been expanded on the plaster model. If the inlay is now cast in the usual manner, it will fit the tooth perfectly, showing that the contraction of the gold has been compensated by the expansion of the

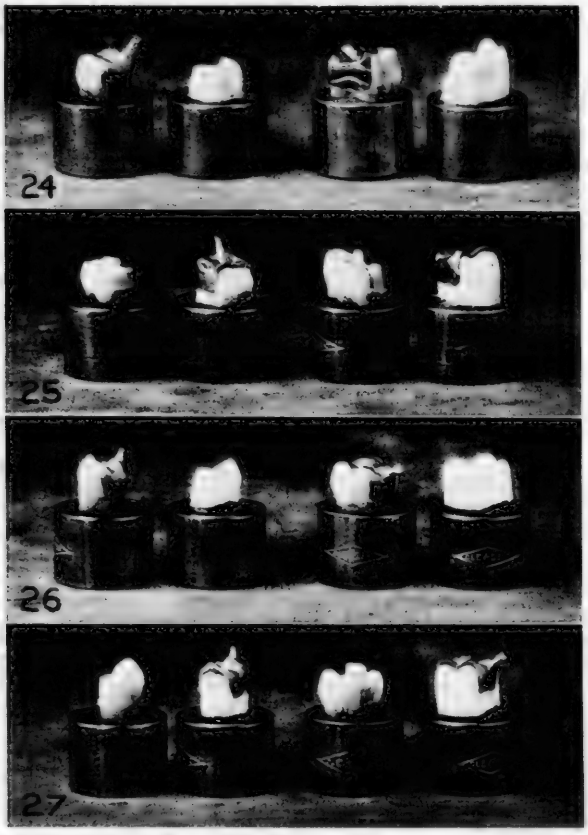


Fig. 24. Two three-fourth crowns with their respective plaster models. See *fig. 25.*

Fig. 25. See *fig. 24.* Shows the contraction of the gold when the cast is on the plaster model.

Fig. 26. Three-fourth and M.O.D. crowns, with their respective plaster models. See *fig. 27.*

Fig. 27. See *fig. 26.* Casts on the plaster models, showing the contraction of the gold.

plaster. We can get an exact idea of the amount of contraction in the gold if we try to put the casting back on the plaster model on which it was made. This is illustrated in *figs. 24* to *31*, inclusive.

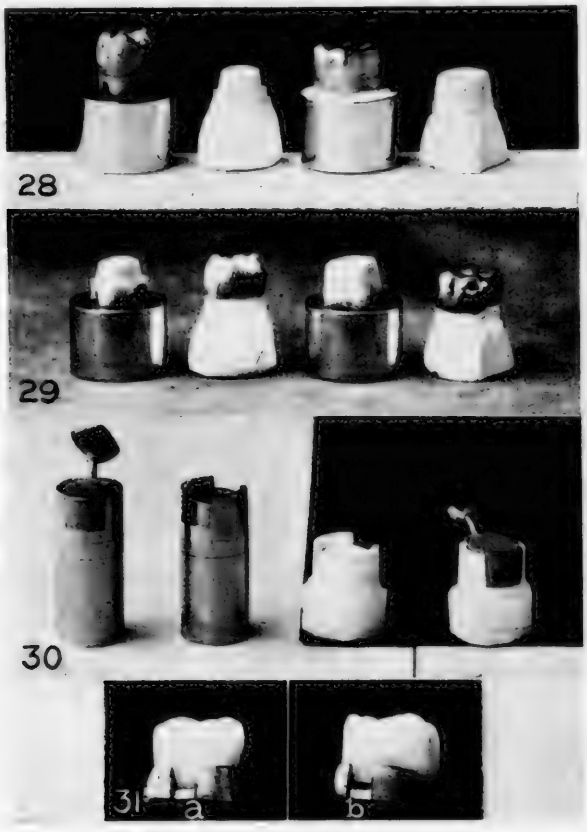


Fig. 28. Two full cast crowns, one with a broad shoulder, the other with the demarcation line. See *fig. 29.*

Fig. 29. See *fig. 28.* Crowns on their plaster models, showing the contraction of the gold.

Fig. 30. Steel die and plaster model, and the same gold casting on each, showing the contraction of the gold on the plaster model.

Fig. 31. Cast gold base for porcelain tooth. *a*—cast after the semi-indirect method. *b*—cast by the usual method.

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CONTENTS

February, 1929

Frederic Austin Peeso. *Arthur N. Cross*..... 3

A critical analysis of the dental literature dealing with the effects of dietary variations upon the structure of teeth. *Henry Klein*, D.D.S..... 5

The relation of nutrition to general immunity. *Sherman L. Davis*, M.D., A.B., A.M., Ph.D..... 11

Biochemical studies of streptococci. I. A comparative study of some common media for the cultivation of streptococci. *Frances Krasnow*, Ph.D., and *William J. Gies*, Ph.D., F.A.C.D..... 29

The bacterial destruction of dental enamel. *Charles F. Bödecker*, D.D.S., F.A.C.D., and *Henry W. C. Bödecker*, B.S., D.D.S., M.D., D.M.D. (Ger.)..... 37

A study of some of the materials used in the casting process. *Oskar Solbrig*, D.D.S..... 55

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